VOLUME 36 NUMBER 2 FEBRUARY, 1953

AMERICAN JOURNAL OF OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

CONTENTS

An anatomic study of the retina ..... *C. C. Teng and H. M. Katzin* 169
Suction as a factor in glaucoma ..... *Adolph Posner* 185
Comparative effects of cortisone ..... *L. Benjamin Sheppard* 193
Nonperforating cyclodiathermy in glaucoma ..... *A. Urrets-Zavalia, Jr., and A. Urrets-Zavalia* 203
Sympathetic ophthalmia following cyclodiathermy ..... *Martin Bodian* 217
DFP in glaucoma ..... *Irving H. Leopold and Albert F. Cleveland* 226
Ocular pemphigus ..... *Peter S. Soudakoff and Harold F. Whalman* 231
Surgical treatment of pemphigus ..... *M. Wallace Friedman and Edwin S. Wright* 237
Pulfrich stereo-effect ..... *Paul W. Miles* 240
Kimmelstiel-Wilson syndrome ..... *Martin Cohen* 243
Cortisone and blood absorption ..... *Walter H. Benedict and Robert W. Hollenhorst* 247
Nystagmus ..... *C. Wilbur Rucker* 250
Pterygium operations ..... *J. William Rosenthal* 259
Acute conjunctivitis ..... *John A. Egan* 260
Hole in macula ..... *W. A. Manschot and C. H. O. M. von Winning* 261
Memory aid for parallax test ..... *Joseph I. Pascal* 262
Electrode for retinal separation ..... *William J. Harrison* 263

DEPARTMENTS

Society Proceedings. 264 Correspondence ... 271 Abstracts 275
Editorials ..... 269 Book Reviews .... 272 News Items 297

For complete table of contents see advertising page xvii

Publication office: 450 Ahnaip St., Menasha, Wisconsin

Copyright, 1953, Ophthalmic Publishing Company, 664 North Michigan Avenue, Chicago 11, Illinois

Subscription price in United States twelve dollars yearly. In Canada and foreign countries fourteen dollars. Published monthly by the Ophthalmic Publishing Company. Subscription and Advertising Office: 664 North Michigan Avenue, Chicago 11, Illinois. Entered as second class matter at the post office at Menasha, Wisconsin. Printed in U.S.A.

Complete ARTIFICIAL EYE *Service*

Experience gained through over 100 years of artificial eyemaking enables us to produce and fit the very finest in artificial eyes. Glass and plastic eyes made to order or available from stock. Eyes fitted to all types of motility implants.

fitting of artificial eyes through the mail

- Orders filled the same day received
- Eyes sent on memorandum
- Liberal assortment of either glass or plastic
- Samples accurately matched
- Superior quality eyes—Finest workmanship

WRITE FOR FREE COLOR CHART AND SIZE GUIDE

CHICAGO
DETROIT
CLEVELAND
KANSAS CITY
MINNEAPOLIS
NEW ORLEANS
ST. LOUIS

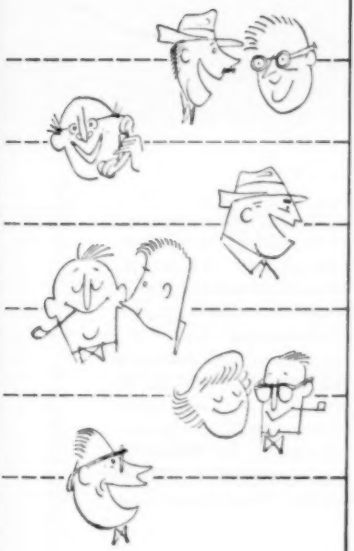
NEW YORK
BALTIMORE
BOSTON
BUFFALO
PHILADELPHIA
PITTSBURGH
WASHINGTON

MAGER & GOUGELMAN, INC.

WHAT THEY'RE SAYING ABOUT THE NEW

Shuron®

RONSIR BROWLINE FRAME...



"It's certainly a winner"

"The best man's frame in the world"

"Perfect for men"

"A hit if I ever saw one"

"Best reaction I've ever had"

"Men go for it"

The Ronsir is only one of the Shuron Browline Family of Fine Frames that are available from Independent Suppliers.

Shuron®

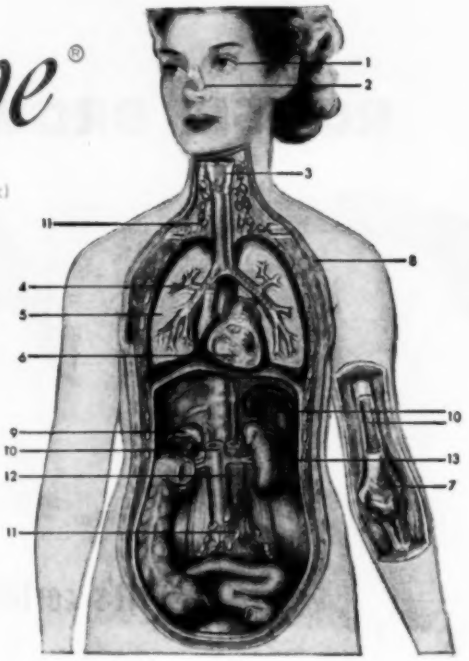
SHURON OPTICAL COMPANY, INC.

GENEVA, N. Y., *Established 1864*

Cortone®

ACETATE

(CORTISONE ACETATE, MERCK)



The many indications for CORTONE *highlight its therapeutic importance in everyday practice*

Primary Site of Pathology and Indications

1. EYE—Inflammatory eye disease. **2. NOSE**—Intractable hay fever. **3. LARYNX**—Laryngeal edema (allergic). **4. BRONCHI**—Intractable bronchial asthma. **5. LUNG**—Sarcoidosis. **6. HEART**—Acute rheumatic fever with carditis. **7. BONES AND JOINTS**—Rheumatoid arthritis; Rheumatoid spondylitis; Acute gouty arthritis; Still's Disease; Psoriatic arthritis. **8. SKIN AND CONNECTIVE TISSUE**—Pemphigus; Exfoliative dermatitis; Atopic dermatitis; Disseminated lupus erythematosus; Scleroderma (early); Dermatomyositis; Poison Ivy. **9. ADRENAL GLAND**—Congenital adrenal hyperplasia; Addison's Disease; Adrenalectomy for hypertension, Cushing's Syndrome, and neoplastic diseases. **10. BLOOD, BONE MARROW, AND SPLEEN**—Allergic purpura; Acute leukemia† (lymphocytic or granulocytic); Chronic lymphatic leukemia.† **11. LYMPH NODES**—Lymphosarcoma†; Hodgkin's Disease†. **12. ARTERIES AND CONNECTIVE TISSUE**—Periarteritis nodosa (early). **13. KIDNEY**—Nephrotic Syndrome, without uremia (to induce withdrawal diuresis). **14. VARIOUS TISSUES**—Sarcoidosis; Angioneurotic edema; Drug sensitization; Serum sickness; Waterhouse-Friderichsen Syndrome.

†Transient beneficial effects.

CORTONE *is the registered trade-mark of Merck & Co., Inc. for its brand of cortisone.*

MERCK & CO., INC.
Manufacturing Chemists
RAHWAY, NEW JERSEY

Schering

CORTOGEN®

ACETATE

for

CORTISONE

therapy

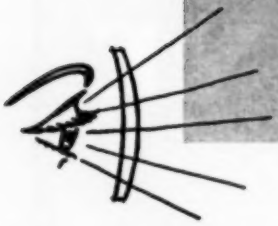
CORTOGEN

The name Schering has come to stand for pioneering research and leadership in steroid hormone chemistry. Now Schering adds this new important product to its steroid line—available in ample amount to meet all your cortisone needs.

Available as 25 mg. tablets, bottles of 30. For complete information write to our Medical Service Department.

Schering CORPORATION • BLOOMFIELD, N. J.

Orthogon Lenses are made the way they are because eyes work the way they do



The image formed by any off-center portion of a lens is degraded by the amount of astigmatic error introduced. This aberration, known as marginal or oblique astigmatism, is one for which the eye can make no compensation or accommodation. And this is the aberration for which the Orthogon series is specifically corrected. Before you prescribe any "corrected" lens, be sure you know *what* it's corrected for. Bausch & Lomb Orthogon is corrected to eliminate the effect of marginal astigmatism—it makes possible clear center-to-edge vision.

In Soft-Lite, too

The Orthogon system of lens correction is based on the principle that only through reduction of astigmatic variation . . . to a level below the physiological limits of human perception . . . can utmost clarity of vision be achieved.

BAUSCH & LOMB Orthogon LENSES

AVAILABLE IN THE WIDEST RANGE OF SINGLE-VISION,

ABSORPTIVE AND MULTI-FOCAL TYPES

1853 1953 INTO A NEW CENTURY OF OPTICAL PROGRESS

BAUSCH & LOMB CENTENNIAL

ALWAYS HAS . . . ALWAYS WILL . . . UNIVIS MAKES THE FINEST OPHTHALMIC PRODUCTS . . . ALWAYS HAS . . . ALWAYS WILL . . . UNIVIS MAKES THE FINEST OPHTHALMIC PRODUCTS . . . ALWAYS HAS . . . ALWAYS WILL . . . UNIVIS MAKES THE FINEST OPHTHALMIC PRODUCTS . . . ALWAYS HAS . . . ALWAYS WILL . . . UNIVIS MAKE

How many arm's length jobs does she do in this room?

She irons, she sets her oven controls, she selects foods—by label—from cupboard shelves, she manipulates the many appliances contrived to save labor. Labor saving—yes. But hard on middle-aged eyes.

Doctors agree it's a mistake to believe that a housewife has no occupational need for intermediate seeing help. There is no doubt that Univis Continuous Vision Lenses—for general and prolonged arm's length seeing—bring new confidence to all presbyopes and greater satisfaction to prescribing doctors.

And all CVs have these things in common: they're made to highest industry standards on the Univis original improved design; no jump as eyes move through all fields; most usable segment area at natural eye level.

Write for Univis Presbyopic Handbook. All CVs insured, of course.

univis THE UNIVIS LENS COMPANY • Dayton 1, Ohio

UNIVIS CVs FOR *ALL* PRESBYOPIC NEEDS

. . . *For Value*

AO NO. 1137 REFRACTING UNIT

Here is a completely hydraulic AO Refracting Unit with handsome chair, upright, lamp bracket and phoroptor arm. Chair and stand are operated independently by sealed oil compression pumps. The chair has soft sponge rubber headrest pads, genuine leather seat and back. Ophthalmometer arm, lamp; round, three-toed, or interlocking base, and other special brackets are optional or may be added later.

The No. 1137 Refracting Unit is supplied in a wide choice of AO standard colors.

AO DELUXE REFRACTING CHAIR

For the very best in refracting chairs you'll choose the AO No. 1106. It offers a complete range of adjustments for size of patient and working position. It may be combined with Junior or Deluxe stands in any suitable arrangement.

Ask your AO Representative about AO Refracting Chairs and Units at your earliest opportunity.

American Optical

INSTRUMENT DIVISION • BUFFALO 15, NEW YORK

KEELER

OPHTHALMOSCOPES

THE INSTRUMENTS WITH THE WORLD-WIDE REPUTATION!

JUNIOR ADJUSTABLE

- Finger-tip controlled focusing collar.
- Inclined lens track gives wide-angle field.
- Red-free filter.
- Macular stop.
- Pre-centered 3v. bulb.
- Lens range plus 36D to minus 42D.
- Illuminated dial.
- Robust featherweight handle, battery or cord (illustrated)

PANTOSCOPE

- Direct or Indirect ophthalmoscopy by polarised, red-free or natural light.
- Intense 12w. light.
- Variable control for projected slit examination of cornea & anterior chamber.
- Wide-angle field with high myopes.
- Lenses plus 30D to minus 50D.
- Illuminated dial with presbyopic viewer.

AUTOFLEX UNIVERSAL UNIT

The all-purpose electrical unit in beautifully polished case. Autoflex cord control on 3v. & 12v. outlets eliminates untidiness. Additional output panel with 3, 6, & 12v. tappings for any instrument taking up to 1 ampere. Rheostat brightness control.

KEELER OPTICAL PRODUCTS, INC.

617 S. 52nd St., Philadelphia 43, Pa.

GRANITE 4-5310

Write or telephone for most up-to-date Brochures.

Acclaimed by Reviewers Everywhere!

Reese's
TUMORS OF THE EYE

By

ALGERNON B. REESE

Attending Ophthalmologist and Pathologist, Institute of Ophthalmology, Presbyterian Hospital; Ophthalmologist to Memorial Center for Cancer and Allied Diseases; Clinical Professor of Ophthalmology, College of Physicians and Surgeons, Columbia University, New York.

584 pages, 511 illus., 122 in full color, $20.00

EXCERPTS FROM A FEW OF THE ENTHUSIASTIC REVIEWS:

- "Occasionally, but only occasionally, a really good book appears in medicine; this is one."

  Stewart Duke-Elder in Brit. M. J.

- "The years of experience of the author in the clinic and pathologic laboratory, the meticulous care reflected on every page, his scientific integrity, assure an accuracy of text that will be difficult to equal and impossible to surpass."

  Derrick Vail in Am. J. Ophth.

- "This book is a comprehensive and authoritative treatise, which will be of very great value. . . . Dr. Reese has combined a detailed account of the pathology of ocular tumors with an analysis of their clinical course richly illuminated by his own vast experience and embellished with sagacious comments in respect to differential diagnosis, surgical techniques, and other therapeutic procedures. The whole is set forth in a fluent and eminently readable text. The illustrations are excellent."

  Arch. Ophth.

- "Exceedingly practical volume for any specialist who may encounter tumors of the eye, its adnexa and their often far reaching complications."

  Bertha A. Klien in Surg., Gynec. & Obst.

- ". . . more than five hundred illustrations are superb in their explanation of the text . . . a more complete and comprehensive group cannot be found anywhere."

  Digest Ophth. & Otolaryng.

YOU MAY ORDER THIS VALUABLE BOOK ON APPROVAL

ON APPROVAL ORDER COUPON

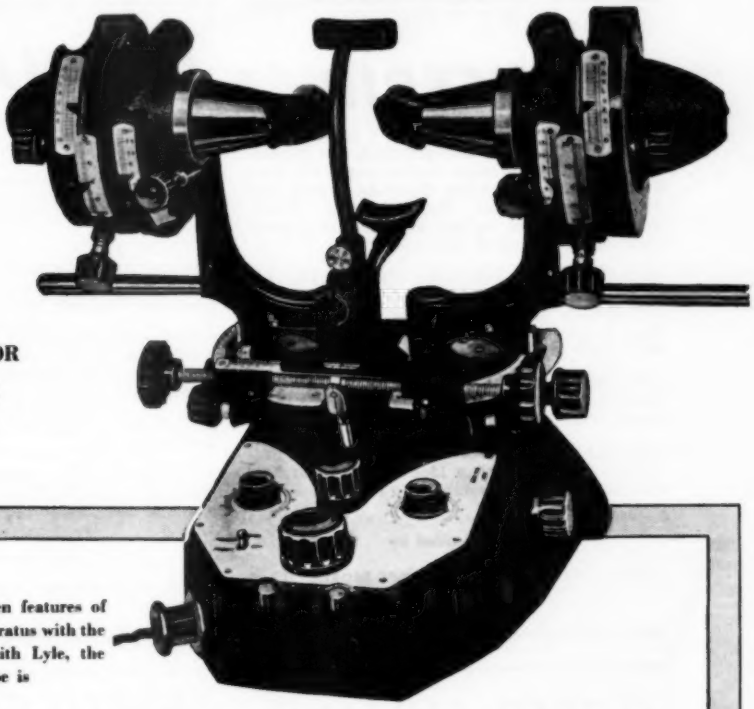
PAUL B. HOEBER, Inc.
Medical Book Department of Harper & Brothers
49 East 33rd Street, New York 16, N.Y.

Send Me ON APPROVAL:

☐ Reese's TUMORS OF THE EYE $20.00
☐ Check Enclosed ☐ Charge My Account

Name .....
Address .....
City ..... Zone ..... State .....

AMJO



THE LYLE MAJOR AMBLYOSCOPE
(Standard Model)

Incorporating the proven features of previous Synotype apparatus with the suggestions of Mr. Keith Lyle, the Lyle Major Amblyoscope is

The Most Advanced Orthoptic Apparatus Produced

Specially manufactured to meet the demands of present day technique, the Lyle Major Amblyoscope, Standard Model, incorporates the following improved features :

1. Elevation and Depression Device
2. Increased range of Duction Control
3. Non-slip Duction Locks
4. Increased Hyperphoria Movement
5. Transparent breath shield
6. Hinged Mirrors and Reflectors
7. Noiseless Slide Carriers
8. Supported main roller bearings and increased mechanical strength
9. Large, easily visible scales
10. Concealed wiring
11. Separate electrical circuits to each optical tube
12. Improved hand flashing switches
13. Provision for automatic moving picture attachment

Specialists in Orthoptic Equipment including :

MOORFIELD'S SYNOPTOPHORE • HESS SCREENS • CHEIROSCOPES • WORTH'S 4 DOT TEST • DIPLOPIA GOGGLES • ROTATING E TEST • MADDOX HANDFRAME • MADDOX WING TEST BAR READERS • ETC.

CLEMENT CLARKE

of England • **63, WIGMORE STREET, LONDON, W.1**

SOLE AGENTS IN U.S.A. CURRY & PAXTON INC. (C) 101 PARK AVENUE, NEW YORK 17. TEL: LEXINGTON 2-7842

ZINCFRIN

DESCRIPTION: A non-stinging, non-irritating ophthalmic solution containing .25% Zinc Sulphate, Benzalkonium Chloride 1:5000 and .12% Phenylephrine Hydrochloride U.S.P. Tonicity and pH values adjusted to closely approximate those of tear secretions.

INDICATIONS: Infectious, allergic, catarrhal or inflammatory conjunctivitis in which the astringency of Zinc Sulphate combined with vaso constrictor properties of Phenylephrine HCl. USP may be desirable.

SUGGESTED DOSAGE: Adult and Child—Two or three drops in affected eye every three hours or as directed by physician.

AVAILABILITY: Zincfrin—Available in prescription pharmacies in 15cc blue dropper bottles. Literature and samples on request.

Manufactured by

ALCON LABORATORIES, INC., FORT WORTH, TEXAS

OP-THAL-ZIN

DESCRIPTION:
A non-stinging, non-irritating ophthalmic solution of Zinc Sulfate ¼% with pH and tonicity values closely approximating those of tear secretions with Benzalkonium Chloride 1:5000 as a "wetting agent" and preservative.

INDICATIONS:
Conjunctival infections in which the astringency of Zinc Sulfate is desirable. Morax-Axenfeld Bacillus, etc.

SUGGESTED DOSAGE:
Adult and Child—Two or three drops in affected eye every three hours, or as directed by Physician.

SUPPLIED: Op-thal-zin 15cc Blue Dropper Bottles.

AVAILABILITY: Available in prescription pharmacies in 15cc blue dropper bottles. Literature and samples on request.

Manufactured by

ALCON LABORATORIES, INC., FORT WORTH, TEXAS

ISOPTO ALKALINE

OPHTHALMIC VEHICLE:
Containing 1% Methyl Cellulose Ophthalmic in aqueous saline solution with Benzalkonium Chloride 1:50,000 as a preservative and "wetting agent."

INDICATIONS:
1. Ophthalmic emollient and lubricating agent in retarded lacrimation states, and as protective lubricant for pathologic conditions.
2. Vehicle for various soluble Ophthalmic Drugs: Antiseptics, Antibiotics, Sulfonamides and various alkaloidal salts.
3. As a physiologic non-greasy suspending agent for insoluble Ophthalmic medications: Cortisone, insoluble Sulfonamides, etc.

ADVANTAGES: Viscosity—plus soothing-lubricating effect—provides better and more prolonged contact of medication with affected area.

AVAILABLE: Isopto Alkaline available in prescription pharmacies in 15cc blue dropper bottles. Samples, literature and suggested prescription formulas available on request.

Manufactured by
ALCON LABORATORIES, INC., FORT WORTH, TEXAS

ISOPTO CETAMIDE

NEW PRODUCT
For increased efficacy of Ocular Sulfonamide therapy—with comfort.
Isopto Cetamide—contains Sodium Sulfacetamide, 15% w/v in soothing .5% Methyl Cellulose Ophthalmic Solution.
INDICATIONS:
Acute chronic conjunctivitis, acute traumatic corneal ulcer, blepharitis and other external eye infections caused by microorganisms susceptible to sulfonamide therapy.

- Comfortable to the eye.
- More intimate and prolonged eye surface contact.
- Better patient cooperation.
- Stable.

CAUTION:
This preparation should be used with care in the treatment of patients who have previously exhibited sensitivity to sulfonamides. Should undesirable reactions occur, discontinue use immediately.
Federal Law prohibits dispensing without a prescription.
Isopto Cetamide is available in 15cc blue dropper bottles in prescription pharmacies. Literature and samples available on request.

Manufactured by
ALCON LABORATORIES, INC., FORT WORTH, TEXAS

Jenkel-Davidson ANNOUNCES

THE CLINI-CAMERA "B"

★ ***Easy, One Hand, "Sight and Shoot" Operation***

★ ***"Sure-Fire" Strobonar Electronic Flash***

★ ***Uses Easily Obtained Standard 828 Film***

★ ***Takes Full Frame One or Two Eye Photos***

★ ***Completely Portable***

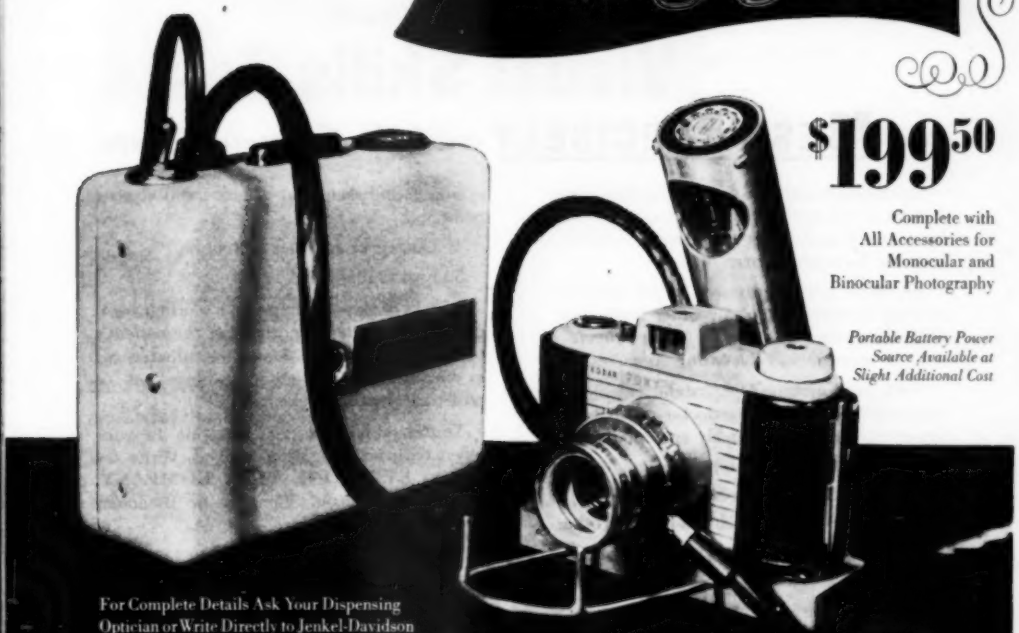
★ ***Lightweight***

★ ***Inexpensive to Operate***

The all new Clini-Camera "B" is the product of years of experimentation, research and actual clinical experience with Jenkel-Davidson's famous Clini-Camera series.

We unhesitatingly guarantee you can take brilliant, color correct external eye photos with the Clini-Camera "B" as easily as you shoot snapshots with a Brownie. No other camera of its type offers so many desirable features.

Jenkel-Davidson
OPTICAL COMPANY



$199.50

Complete with All Accessories for Monocular and Binocular Photography

Portable Battery Power Source Available at Slight Additional Cost

For Complete Details Ask Your Dispensing Optician or Write Directly to Jenkel-Davidson

366 POST STREET, SAN FRANCISCO 19, CALIFORNIA

can MESH PRECISELY with the Screening Tests

Ophthalmologists are simplifying the examination of referred patients, and saving time, by using Visual Skills tests that *coordinate with the Screening Tests.*

Keystone Screening is used by *far more Industrial Concerns* than any other method or instrumentation—also by *far more schools*—and *far more Driver Licensing Agencies.*

With the Keystone Ophthalmic Telebinocular and Skills Tests, the doctor or his technician can determine quickly and accurately:

1. Presence or absence of hyperphoria.
2. Direction and degree of heterophoria.
3. Usable Vision in each eye with the other eye open and seeing, at distance and at near point.
4. Degree of macular stereopsis.
5. Fusion readiness.

Checks of these findings, on a scoring sheet, give you an immediate *profile of the patient's pattern of visual functioning*—a valuable aid in diagnosis and refraction; also excellent for post-treatment recheck.

You may have a demonstration in your office with no obligation implied. Write for details. **KEYSTONE VIEW COMPANY, Meadville, Penna.** Pioneers in Binocular Testing Equipment.

KEYSTONE VISUAL SKILLS TESTS

PRESCRIPTION OPTICIANS

ST. LOUIS, MO.

Erker Bros. Optical Co.

610 Olive Street

518 N. Grand Boulevard

and Clayton, Mo.

Prescription Opticians Since 1879

CHICAGO, ILL.

ALMER COE & COMPANY

Prescription Opticians

Established 1884

10 N. Michigan Ave.

1645 Orrington Ave., Evanston, Ill.

Dow Optical Co.

PRESCRIPTION SPECIALISTS

Suite 1015 30 N. Michigan Avenue

Chicago, Illinois

Phone RAndolph 6-2243-44

DEALERS IN OPHTHALMOLOGICAL EQUIPMENT

PORTLAND, ORE.

Hal. H. Moor, 315 Mayer Bldg.

Guild Optician

Oculists' prescriptions exclusively

NEW YORK CITY

E. B. Meyrowitz

INCORPORATED

Optician Established 1875

520 Fifth Ave., New York

255 Livingston St., Brooklyn

Member Guild of Prescription Opticians of America

CINCINNATI, OHIO

L. M. Prince Co.

Established 1872

Prescription Opticians

Sole makers of Coflexic

Corrected Curve Lenses

storz Surgical Instruments . . . NEW!

Two

Vitreous Foreign Body FORCEPS

328bc

328b* Forceps, Vitreous Foreign Body, THORPE: with delicate tip for the removal of irregular foreign bodies.

328c* Forceps, Vitreous Foreign Body, THORPE: designed especially for the removal of lead shot. Instrument is inserted with blades closed, and should not be allowed to open until foreign body is reached.

Both Stainless Steel, each $16.50

Shown in our 1953 Supplement to Eye Instrument Catalog. Copies sent upon request.

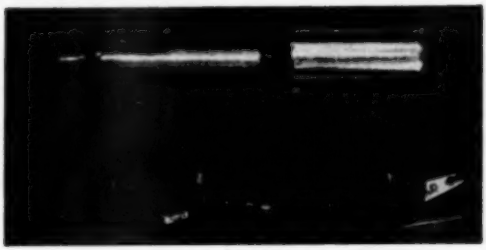
storz

Storz Instrument Company

4570 Audubon Ave., St. Louis, Missouri

GREEN'S ELECTRIC TREPHINE

IMPROVED MODEL BY DAVID KADESKY, M.D.



The instrument simplifies the operation of trephining for Glaucoma or Corneal Transplantation. Blades are provided with a shoulder that prevents penetration beyond 1.1 mm., and are available in 1½ mm., 4.5, 4.6, 5.5, 5.6, 6.5, 6.6, mm. diameters.

Trephine, case, and two 1½ mm. blades $95.00

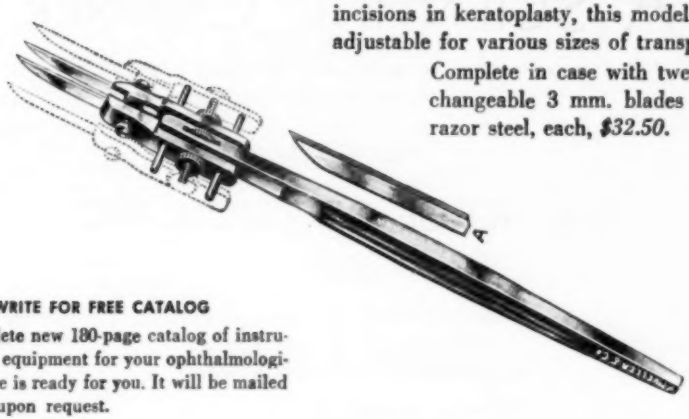
PARSONS OPTICAL LABORATORIES, INC.

518 Powell Street San Francisco 2, Calif.

Castroviejo's—TWIN KNIFE FOR KERATOPLASTY

Most useful for marking simultaneous parallel incisions in keratoplasty, this model is easily adjustable for various sizes of transplants.

Complete in case with twelve interchangeable 3 mm. blades of finest razor steel, each, *$32.50.*



WRITE FOR FREE CATALOG

Our complete new 180-page catalog of instruments and equipment for your ophthalmological practice is ready for you. It will be mailed promptly upon request.

ORDER DIRECT FROM

V. Mueller and Company

330 SOUTH HONORE STREET
CHICAGO 12, ILLINOIS

THE AIMARK PROJECTION PERIMETER

A FINE INSTRUMENT FOR FIELD EXAMINATION AND DIAGNOSIS

Price $675.00 f.o.b. New York
Complete with Transformer

One of many superb British-made ophthalmic products, the result of a long tradition of expert design and skilled craftsmanship, imported by

CURRY & PAXTON, INC.
101 Park Avenue, N.Y. 17, N.Y.

from distinguished instrument makers:
ALLIED INSTRUMENT MFRS., LTD.
CLEMENT CLARKE, LTD.
CURRY & PAXTON, LTD.

Write for Catalog—Price List J

Distributed in this country by outstanding optical firms, including:
New York: E. B. Meyrowitz Co. Chicago: The House of Vision, Inc.
And at the Park Avenue Showrooms in New York

A SCIENTIFIC CORNER

WHERE OPTICAL PROBLEMS ARE DISCUSSED AND THE SOLUTIONS, AS SOLVED BY EXPERIENCE, INDICATED!

PRESENTED BY

THE HOUSE OF VISION, INC.

CHICAGO

EVANSTON
DES MOINES
HIGHLAND PARK
MASON CITY
MUSKEGON
OAK PARK
MILWAUKEE
SIOUX CITY
AURORA
MINNEAPOLIS

DISTANCE CENTERS AND VERTICAL IMBALANCES

If the induced imbalances in bifocal segments are small, that is, .7 prism diopters or less, changing the position of the centers of the distance lenses will effect a compromise and give your patient good balance.

Let us assume a case that requires a +1.00 sphere in one eye and +0.25 sphere in the other eye. This combination of corrections will result in an induced imbalance of .6 prism diopters.

If the optical centers of the distance lenses are ordered positioned at the tops of the bifocal segments, the near imbalance will be reduced to .37 and you will have introduced a .22 error in the distance lenses. These errors are so small they will cause no trouble.

This method of correcting small induced imbalances has proved very satisfactory. Always, of course, it is at the discretion of the ophthalmologist as to whether or not these tiny imbalances need correction.

"IF IT'S A LENS PROBLEM, LET'S LOOK AT IT TOGETHER"

AMERICAN JOURNAL OF OPHTHALMOLOGY

SERIES 3 · VOLUME 36 · NUMBER 2 · FEBRUARY, 1953

CONTENTS

ORIGINAL ARTICLES

An anatomic study of the peripheral retina: III. Congenital retinal rosettes. C. C. Teng and H. M. Katzin 169
Suction anterior to the iris as a factor in narrow-angle glaucoma. Adolph Posner 185
Comparative effects of cortisone on rabbit eyes treated by cyclodiathermy and cyclo-electrolysis. L. Benjamin Sheppard 193
Nonperforating cyclodiathermy in the treatment of glaucoma: Diathermia retrociliaris. A. Urrets-Zavalía, Jr., and A. Urrets-Zavalía 203
Sympathetic ophthalmia following cyclodiathermy. Martin Bodian 217
Di-isopropyl fluorophosphate (DFP) (0.01 percent) in chronic wide-angle glaucoma. Irving H. Leopold and Albert F. Cleveland 226
Ocular pemphigus: Report of a case with the histologic findings in the cornea. Peter S. Soudakoff and Harold F. Whalman 231
The surgical treatment of ocular pemphigus. M. Wallace Friedman and Edwin S. Wright .. 237
The Pulfrich stereo-effect produced by monocular magnification without reducing illumination. Paul W. Miles 240
Kimmelstiel-Wilson syndrome: Review of the literature and report of a case with ophthalmoscopic findings. Martin Cohen 243
The influence of cortisone on the rate of blood absorption from the eye. Walter H. Benedict and Robert W. Hollenhorst 247
Nystagmus. C. Wilbur Rucker 250

NOTES, CASES, INSTRUMENTS

The Blaskovicz pterygium operation. J. William Rosenthal 259
Acute conjunctivitis with lymph-gland suppuration. John A. Egan 260
Hole in the macula due to syphilis: Report of a case. W. A. Manschot and C. H. O. M. von Winning 261
Memory aid for the parallax test. Joseph I. Pascal 262
An electrode for use in retinal separation. William J. Harrison 263

SOCIETY PROCEEDINGS

Colorado Ophthalmological Society, September 20, 1952 264
Chicago Ophthalmological Society, March 17, 1952 265
Ophthalmological Society of Madrid, June 6, 1952 267

EDITORIALS

Herpes corneae 269
International Council of Ophthalmology 270

XVII CONGRESS OF OPHTHALMOLOGY 271

CORRESPONDENCE

Retrolental fibroplasia in Negroes 271

BOOK REVIEWS

Biomicroscopie et Histopathologie de l'Oeil 272
Transactions of the French Ophthalmological Society 273
Archivos de la asociación para evitar la ceguera en Mexico 273

ABSTRACTS

General pathology, bacteriology, immunology; Vegetative physiology, biochemistry, pharmacology, toxicology; Physiologic optics, refraction, color vision; Diagnosis and therapy; Ocular motility; Conjunctiva, cornea, sclera; Uvea, sympathetic disease, aqueous; Glaucoma and ocular tension; Crystalline lens; Retina and vitreous; Optic nerve and chiasm; Neuro-ophthalmology 275

NEWS ITEMS 297

"rapidly gaining popularity" in E.E.N.T. infections

Sulfamylon® 5%

HIGHLY EFFECTIVE...

...WELL TOLERATED

"**Sulfamylon** is rapidly gaining popularity in the local treatment of ear, nose and throat infections as a very effective bactericidal and bacteriostatic agent."[1]

Sulfamylon is "consistently effective in vitro against the organisms commonly found in otitis externa."[2]

"**Sulfamylon** is an effective local therapeutic agent in external otitis."[1]

"**5% Sulfamylon** in methyl cellulose is a valuable weapon for the therapist dealing with external otitis."[4]

Sulfamylon 5% aqueous solution, bottles of 1 fl. oz. and 8 fl. oz.; 5% viscous solution, bottles of 1 fl. oz.
Sulfamylon 5% aqueous solution, bottles of 100 cc., with vial of Streptomycin 20,000 units.

WINTHROP-STEARNS

Sulfamylon, trademark reg. U. S. & Canada, brand of mafenide

1. Diehl, K. L.: *Arch. Otolaryng.*, 55:68, Jan., 1952.
2. McLaurin, J. W.: *Laryngoscope*, 61:66, Jan., 1951.
3. Craig, P. E.: *Jour. Oklahoma Med. Assn.*, 42:523, Dec., 1949
4. Lott, James E.: *Air University, USAF School of Aviation Medicine*, Report No. 1, Project No. 21-32-026, p. 4.

WINTHROP-STEARNS INC. • NEW YORK 18, N. Y., WINDSOR, ONT.

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 36 FEBRUARY, 1953 NUMBER 2

AN ANATOMIC STUDY OF THE PERIPHERAL RETINA*

III. CONGENITAL RETINAL ROSETTES

C. C. TENG, M.D., AND H. M. KATZIN, M.D.

New York

Pursuing our study of the peripheral retina, we wish to report on a group of lesions that fall into the category of rosettes. The study was made, as before, on Eye-Bank eyes. After the cornea was removed for surgical purposes, the eyes were split in half and studied in a fluid chamber with the slitlamp. Lesions were drawn and marked so that serial histologic sections could be made.

Rosettes have been described rather extensively as they occur in retinal tumors; a few authors have described them as a rare condition in both normal and abnormal eyes.

The incidence of rosettes in normal eyes, particularly in infants' eyes, is quite high when the lesions are sought for in such a manner that the entire circumference of the retinal periphery can be carefully examined.

Several distinct types of rosettes have been found in our specimens, and we have studied the relationship of rosettes to the granular patches described in a previous paper.[1]

REVIEW OF LITERATURE

Seefelder,[2] 1909, was the first to describe rosettes in the region of the optic nerve and retina in three normal fetal eyes. He emphasized the importance of the study in connection with retinal tumors.

Lindenfeld,[3] 1913, described similar findings in normal fetal eyes and also reported that X rays can produce the formation of rosettes in fetal eyes.

Goldstein and Wexler[4] confirmed the effect of X rays in causing rosette formation in rabbit experimentation and also in human cases. Their paper includes an excellent review of the literature.

Fuchs,[5] in 1917, described folds, rosettes, cysts, and irregular proliferation of the nuclear layers of the retina as congenital defects in children and adults.

Jaensch,[6] 1925, reported rosette formation in microphthalmos. He speculated that rosettes might serve as a starting point for gliomas.

Joy[7] reported rosette formation in an eye with a coloboma of the choroid, and Delaney[7] and Gartner[8] in cyclopia.

Pusey and Brown[9] reported rosette formation in inflammatory processes of the eye. Murakami[10] observed rosette formation in a case of disseminated choroiditis.

INCIDENCE

Out of 40 children, 72 eyes were examined and there were rosettes in 24 eyes of 17 children three of whom were premature, nine were less than one year of age at death, and seven were up to seven years of age at death. From one to 13 rosettes were found in a single eye.

Retinal folds were found in only one child but in both eyes. Folds of the nonpigmented epithelium were found in 12 eyes out of 23. They were only seen in eyes which also contained rosettes.

* Under the auspices of The Eye-Bank for Sight Restoration, and supported in part by research grant B-153 from The National Institute of Neurological Diseases and Blindness of the National Institute of Health, Public Health Service.

The nasal side was most frequently involved, all 23 eyes with rosettes being involved on the nasal side; four of these eyes also had rosettes on the temporal side. Of the folds, 12 occurred nasally and two temporally.

The details are outlined in Table 1. In none of the eyes were the lesions so extensive as to remove the eye from the category of normal.

Pathology

Three main types of rosettes were distinguished:

A. Rosettes occurring in congenital folds of the nonpigmented epithelium of the pars plana.

TABLE 1

Summary of data in cases showing rosettes

	Eye No.	Eye	Age	Sex	No. of Rosettes*		No. of Folds†		Cause of Death
					Nasal	Temp.	Nasal	Temp.	
1	1768	OS	Premature	F	6	—	3	—	Prematurity
	69	OD			1	—	1	—	
2	1835	OS	Premature	F	2	—	1	—	Prematurity
	36	OD	7½ mo.		—	—	—	—	
3	1898	OD	Premature	F	1	—	1	—	Prematurity
	99	OS	(destroyed)						
4	2076	OD	Stillborn	M	1	—	—	—	Asphyxia with retinal hemorrhage
	77	OS			1	—	—	—	
5	1787	OS	Full-term	F	3	3	1	—	Birth injury
	88	OD	(destroyed)						
6	1880	OS	1 day	M	2	1	3	1	Atelectasis of lungs††
	81	OD			2	1	2	2	
7	1870	OS	2 days	M	—	—	—	—	Congenital heart
	71	OD			1	—	—	—	
8	1950	OS	2 wks.	F	12	—	4	—	Respiratory infection
	51	OD			6	—	4	—	
9	2137	OD	5 mo.	F	—	—	5	—	Miliary tuberculosis
	38	OS			1	—	1	—	
10	2057	OD	(destroyed)						
	58	OS	2 yr.	M	10 (two rosettes with a tendency to detach or form floaters)†††	—	2	—	Urinary infection
11	2158	OD	2 yr.	M	1 (plus one undetached floater)†††	—	—	—	Skull fracture and retinal hemorrhage
12	2246		2 yr.	F	2 (1 undetached floater††† with vitreous adhesion)	1	—	—	Obstruction of common bile duct
13	2185	OS	3½ yr.	F	13	—	—	—	Leukemia
	86	OD		F	—	—	—	—	
14	2019	OS	5 yr.	M	1 (in form of undetached floater†††)	—	—	—	Infectious disease
	20	OD			—	—	—	—	
15	2096	OD	5½ yr.	M	1 (in form of undetached floater†††)	—	1	—	Third degree burns
	97	OS			5	—	—	—	
16	1703	OS	7 yr.	M	—	—	—	—	Status epilepticus
	04	OD			2 (one of the two floaters with string attachment to the retina†††)	—	—	—	
17	2079	OS	Unknown	F	1	—	1	—	Congenital heart

* = The rosettes had their origin from congenital remnants of retinoblasts.
† = The folds consisted of nonpigmented epithelium.
†† = The case with retinal folds.
††† = In these cases the cell mass had begun to detach.

B. Rosette formation from congenital remnants of retinoblasts.

C. Rosettes in congenital retinal folds.

GROUP A

The folds of nonpigmented epithelium are found in irregular patches or long narrow ridges over the ciliary process, pars plana, ora, or the retinal periphery. They are always arranged in a meridional direction. From the age incidence, it may be inferred that the folds peripheral to the ora tend to disappear at about the time of birth. Those found in the retina persist and may remain throughout life.

The appearance of these folds by slitlamp varies with age. In the infant, the surface is smooth, although the outline may not be perfectly regular. In older eyes, the surface of the fold becomes granular because of the formation of vitreous adhesions and the development of cystoid degeneration. All or part of the fold may become detached to form a vitreous floater.

Histologically, in infants' eyes, the characteristic folding of the nonpigmented epithelium is easily made out.

Fusion may occur irregularly between cell layers in the region of the fold, and then rosettes are formed. These are usually tubular or oval and consist of a single layer of nonpigmented epithelium. When a fold of nonpigmented epithelium overlies the retina, the layer in contact with the retina proliferates and differentiates forming retinal tissue.

GROUP B

Rosette formation from congenital remnants of retinoblasts usually occurs only in the peripheral retina. In all our specimens rosettes were found within three millimeters of the ora. Several cases reported in the literature, however, described them as far back as the optic disc. We have found them singly or in rows of two or three, arranged in a meridional fashion at the base of the ora. They are not infrequently found in line with congenital folds of the nonpigmented epithelium.

The intervals between multiple lesions vary considerably. The size of the lesions ranges from 20 microns to one mm. in diameter; the smaller ones appear as opaque dots; the larger ones appear ring-shaped. The larger ones usually are found more posteriorly.

With the slitlamp, rosettes are seen to be spherical or oval in outline; they have a smooth surface in babies' eyes, granular in adults. The larger part of the lesion is embedded in the retina, but may protrude somewhat into the vitreous. Some, even in infants, show a marked tendency to form floaters. Histologically, rosettes formed from retinoblastic remnants are classified into four subtypes:

Type 1. Fully differentiated cells without proliferative activity

These rosettes are very similar to rosettes in neurocytoma. All the cells resemble fully differentiated retinal cells. The rosettes are found somewhere between the internal limiting membrane and the external nuclear layer, originating from the latter.

There is a cavity in the center, lined with a basement membrane that resembles and may appear to be continuous with the external limiting membrane of the retina. As evidence of the cellular maturity of these rosettes, bipolar cells surround them.

The same changes that occur in the peripheral retina may be seen in these rosettes. Cystoid degeneration occurs in either the internal or external walls of rosettes, often at a much younger age than in the normal retina. The lesions take on a granular appearance in later life because of peripheral cystoid degeneration.

Vitreous adhesions develop at an early age and may lead to floaters as well as retinal holes. Those lesions that remain in contact with the vitreous for long periods tend to undergo cellular changes so as to resemble the nonpigmented epithelium of the ciliary body.

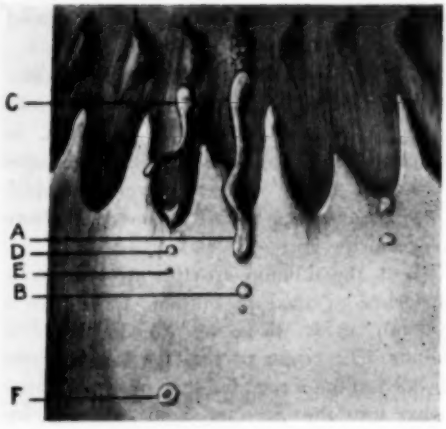


Fig. 1 (Teng and Katzin). *Case 1769.* Female premature infant, left eye, nasal side. (Complete description in text.)

Type 2. Less differentiated cells with signs of active proliferation

These lesions are on the average much smaller than the better differentiated ones and are situated more peripherally. They consist of neuroblasts with elongated cell bodies and very little cytoplasm, and are composed chiefly of the elongated nucleus packed with densely and deeply staining chromatin. A basement membrane is not always present. Few or no bipolar and ganglion cells surround the lesion.

Type 3. Frankly proliferating retinoblasts arising from the external nuclear layer

These cell masses have no cavity and no basement membrane. They are seen to be migrating inwardly from their origin in the external nuclear layer. No bipolar or ganglion cells accompany the mass, which proliferates in a direction perpendicular to the surface of the retina. Most small vitreous floaters found near the periphery of the retina are of this origin. They may appear as young shoots or buds.

Type 4. A fourth type is seen which is similar to Type 3

This is usually a purely histologic finding, being too small to be seen by slitlamp. They are very common and consist of only a few cells.

Groups A and B

The following cases have been selected to illustrate congenital folds of nonpigmented epithelium and rosettes from remnants of retinoblasts (Groups A and B).

Case 1768-9

A female premature infant died 15 hours after birth. Both eyes are normal except for the following findings:

Right eye. The nasal side of the right eye has one small fold at the margin of the ora and a small rosette in the same meridian about one mm. from the ora. The temporal side of this eye is normal.

Left eye (fig. 1). On the nasal side, there are three irregular folds. Two extend from the ciliary body to the anterior portion of the retina. The third is a small fold at the margin of the ora. These are composed of nonpigmented epithelium from the pars plana. Posterior to A (fig. 1) is a small rosette, B. Represented by C is an irregular fold of nonpigmented epithelium over the

Fig. 2 (Teng and Katzin). Microscopic section of fold (A) of Figure 1.

ciliary process and pars plana. D indicates a Type 2 rosette just behind the ora serrata; E is a Type 3 rosette and F a Type 1 rosette. In addition, irregular active proliferative cell centers are found scattered in a region of the ora. These are Type 4 rosettes.

Microscopic section of fold A in Figure 1 is shown in Figure 2. Because of the overhanging double layer of nonpigmented epithelium arising from the pars plana, three layers exist, counting the retina. The two layers of nonpigmented epithelium do not readily fuse, but the layer in contact with the retina begins to form retinal tissue, Figure 3. When the two layers of nonpigmented epithelium do fuse, rosettes are formed which are of the medullo-epithelioma type, Figure 4.

Fig. 3 (Teng and Katzin). High magnification of Figure 2, showing proliferation, differentiation, and migration of nonpigmented epithelial cells of the fold adjoining the retina.

. Fig. 4 (Teng and Katzin). Section from (C) of Figure 1. This type of rosette forms from folding of nonpigmented epithelium.

A typical Type 1 rosette, section F of Figure 1, is seen in Figures 5 and 6. Note the well-developed basement membrane and the surrounding bipolar cells.

In Figures 7 and 8 are shown a typical Type 2 rosette sectioned from D of Figure 1. Note the origin from the external nuclear layer, the lesser differentiation of the cells, and the basement membrane. There are a few bipolar cells surrounding it.

In Figures 9 and 10 is another example of a Type 2 rosette but at an earlier stage. This illustration would suggest a continuity between the external limiting membrane of the retina and the basement membrane of the rosette. However, further study does not bear this out.

In Figure 11 is a Type 3 rosette sectioned from E of Figure 1 and Figure 12 is a Type 4 rosette which was not located by slitlamp study of the gross specimen. These have no cavity or basement membrane. Their cell types indicate immaturity.

CASE 1950

A female infant, aged two weeks, died of respiratory infection.

Left eye (nasal side, fig. 13). At the edge of the ora and posterior to it are five folds and six rosettes posterior to them. The largest rosette is ring-formed.

Figure 14 shows a section through G of Figure 13. The lesion at the ora is a fold of nonpigmented epithelium (with a rosette incorporated) composed of a single layer of nonpigmented epithelium. The adjoining lesion is also a fold, probably a portion of the first lesion. Further posteriorly is a Type 1 rosette in which part of the basal membrane can be made out.

Fig. 5 (Teng and Katzin). Section from (F) of Figure 1. A typical Type 1 rosette.

CASE 1787

A female newborn infant died of birth injury.

Left eye. Among six rosettes present only one is Type 1 (fig. 15). This differs from the previous examples in two respects: (1) Cystoid degeneration is found inside the cyst, and (2) there are local vitreous adhesions indicating that such changes can develop before birth.

CASE 2058

A two-year-old boy died of urinary infection.

Left eye. Temporal side, normal. On the nasal side are two small folds and 13 rosettes (fig. 16).

Two of the lesions illustrate proliferation of Type 3 or 4 rosettes anteriorly over the pars plana, represented by H. Two of the rosettes protrude into the vitreous and have clear cystoid spaces at the base, represented by I.

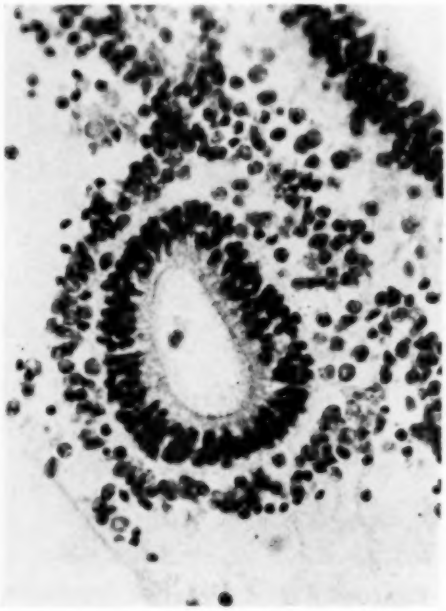


Fig. 6 (Teng and Katzin). High magnification of the Type 1 rosette in Figure 5.

Fig. 8 (Teng and Katzin). High magnification of the Type 2 rosette in Figure 7.

Figure 17 shows detailed section from lesion H of Figure 16.

Figure 18 shows detailed section from lesion I of Figure 16.

Cystoid change in the retina at the base of the rosette is the first step in the development of floaters.

CASE 2019

A five-year-old boy died of chickenpox encephalitis.

Right eye (nasal side, fig. 19). The gross findings are normal except for a spherical mass 50 microns in diameter with a short stem attached to the retina. The lesion is about 0.3 mm. from the ora. The surface of the mass is relatively smooth. Histologically the lesion is an undetached floater without

Fig. 7 (Teng and Katzin). Section from (D) of Figure 1. A typical Type 2 rosette.

Fig. 9 (Teng and Katzin). Section from (B) of Figure 1. A typical Type 2 rosette, but less differentiated than the one shown in Figures 7 and 8.

change in the retina. The cells resemble nonpigmented epithelium. There are fibrillar attachments to the retina, and local adhesions to the vitreous.

It is a Type 2 or 3 rosette.

The three cases just presented illustrate the development of floaters from rosettes in baby eyes. They are very similar to those we

Fig. 10 (Teng and Katzin). High magnification of the Type 2 rosette in Figure 9.

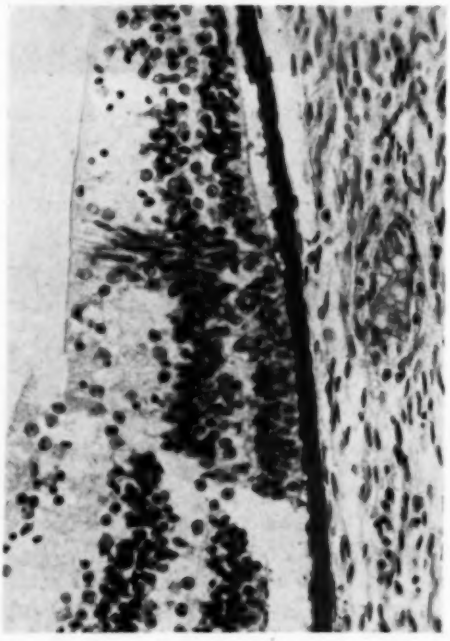


Fig. 11 (Teng and Katzin). Section from (E) of Figure 1. A typical Type 3 rosette.

Fig. 12 (Teng and Katzin). Type 4 rosette, showing irregular remnants of retinoblasts.

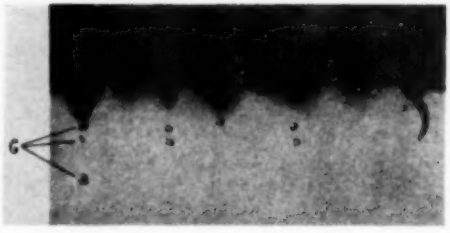


Fig. 13 (Teng and Katzin). *Case 1950.* A female infant, aged two weeks, left eye, nasal side.

have seen in the older age group which we have reported in a previous paper.[1] They form vitreous adhesions and are associated with local cystoid degenerative changes. Such floaters may occur as early as the age of three and one-half years. The larger rosettes separate in later life and may leave a hole. Smaller ones separate more easily, are more common, and leave only a small degenerated spot in the retina.

Fig. 14 (Teng and Katzin). Section from (G) in Figure 13, showing two folds and a Type 1 rosette.

GROUP C

Rosettes in congenital retinal folds are illustrated by these cases.

CASE 1880

This infant died of atelectasis at birth. Out of all the eyes examined, only this pair (both eyes) has congenital retinal folds. Both eyes are normal except for the folds.

Right eye (temporal side, fig. 20). The lesion is a thin fold of tissue, continuous

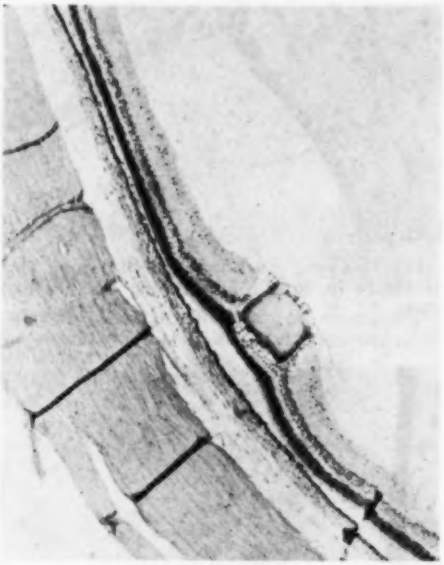


Fig. 15 (Teng and Katzin). *Case 1787.* A newborn female infant, left eye, nasal side. This shows a Type 1 rosette with local vitreous adhesion and cystoid degeneration in the wall of the cyst.

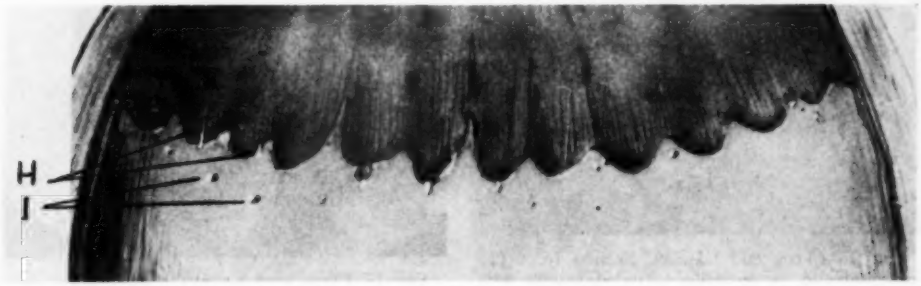


Fig. 16 (Teng and Katzin). *Case 2058.* A boy, aged two years, left eye, nasal side. At (H) two rosettes are proliferating anteriorly over the pars plana. At (I) two rosettes show cystoid change at the base.

with the retina. The small irregularities in the folds represent rosettes. On the nasal side of the eye is a similar lesion.

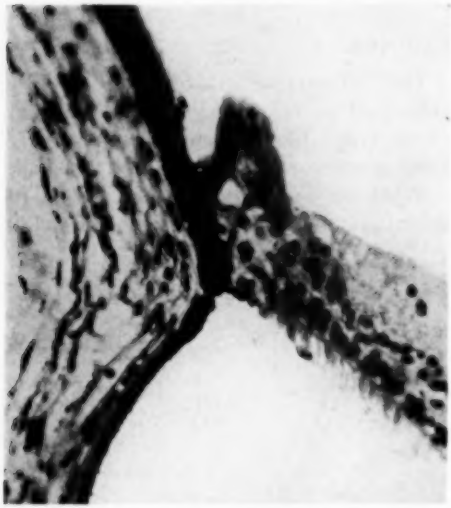


Fig. 17 (Teng and Katzin). Section from (H) of Figure 16. Rosette proliferating over the pars plana.

Figure 21 is a histologic section from Figure 20. The membrane is thinned-out retina extending from the edge of normal retina to the pars plana. The cells of the membrane are similar to those in the external nuclear layer and there are no inner nuclear layers or ganglion cell layers. The external limiting membrane of the normal retina continues to the edge of the abnormal area, then suddenly stops.

Many rosettes are seen in the thinned-out retina. Most of them are oval in form, one is tubular. A basal membrane is seen in each of the rosettes and from one to four rows of cells surround the lumen, including some bipolarlike cells. These rosettes strongly resemble those seen in neuro-epithelioma.

Figure 22 is a high-power section of Figure 20. Several rosettes are represented. Figure 23 is a high-power section from Figure 20. A rosette close to the ora con-

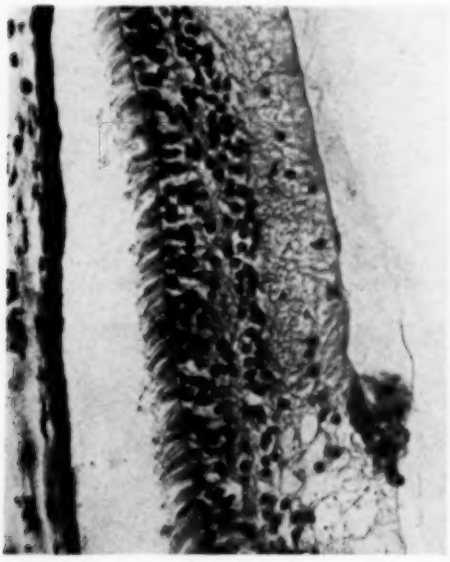


Fig. 18 (Teng and Katzin). Section from (I) of Figure 16. A partially extruded rosette with adhesion to the vitreous and cystoid degeneration at its base.

Fig. 19 (Teng and Katzin). *Case 2019.* A boy, aged five years, right eye, nasal side. Undetached floater from a rosette (Type 2 or 3).

tains nonpigmented epithelial cells on the side of the vitreous. The side in contact with retina is proliferating and differentiating into retinal cells.

CASE 1881

Left eye (fig. 24). The main lesion contains examples of three types of rosettes—A, B, and C—rosettes formed from folding of nonpigmented epithelium, from remnants of retinoblasts, and from retinal folds. The adjoining ciliary processes are hypertrophied.

Figure 25 shows a section through the main lesion of Figure 24. Folded nonpigmented epithelium replaces the retina which comes to an end one mm. from the ora. A few rosettes are found in the fold, of the medullo-epithelioma type.

Figure 26 is a higher magnification of Figure 25 and shows clearly the proliferation and differentiation (metaplasia) of the nonpigmented epithelium where it comes in contact with retina.

Figure 27 is a high-power section of Figure 25. The external limiting membrane

Fig. 21 (Teng and Katzin). Low magnification of a section from Figure 20, showing the congenital retinal fold with a number of rosettes.

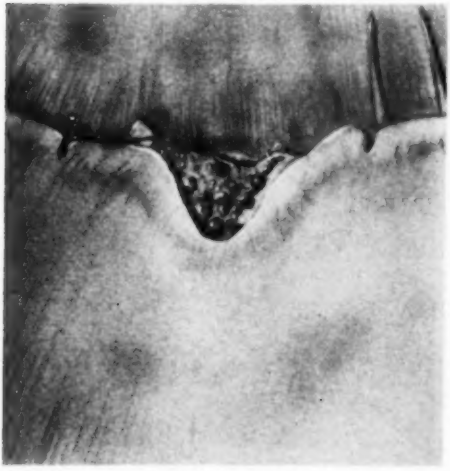


Fig. 20 (Teng and Katzin). *Case 1880.* A newborn male infant, right eye, temporal side. Gross picture of rosettes in a congenital fold of the peripheral retina.

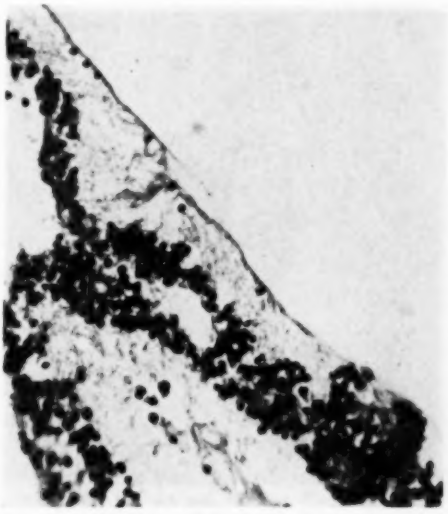


Fig. 22 (Teng and Katzin). High magnification of the retinal fold with a few rosettes (neuroepithelioma type).

of the retina comes to an end before the region of the fold.

A rosette can be seen (neuro-epithelioma type) which has a basement membrane within.

The special significance of Cases 1880 and 1881 are:

In Case 1880 there is a retinal fold, and in Case 1881, in addition to a retinal fold, there is a break in the retina which is replaced by a fold of nonpigmented epithelium arising from the pars plana.

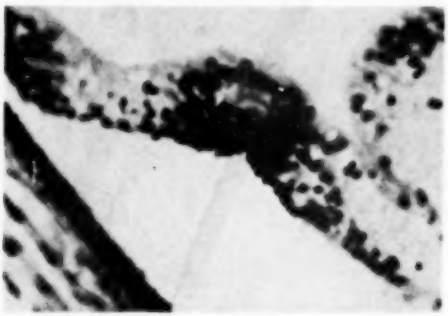


Fig. 23 (Teng and Katzin). Detail from Figure 20. Note the rosette close to the ora. On the vitreous side the cells resemble nonpigmented epithelium, on the retinal side they are undergoing metaplasia into retinal cells.

In both eyes the normal development of the retina is disturbed at the site of the lesion. The retina is thin and there is no external limiting membrane. Proliferating retinoblasts form rosettes and may also proceed to form a basement membrane as evidence of differentiation. To date we have not found any instance in which the basement membrane of a rosette is continuous with the external limiting membrane of the retina, so that we believe the rosettes are formed independently and not by budding or branching.

Discussion

I. Mechanism of rosette formation

Three kinds of congenital anomalies produce rosettes:

1. Folding of nonpigmented epithelium of the pars plana over the retina

The rosettes form where fusion occurs between adjoining layers of the fold. These rosettes are tubular or oval, and derive their appearance from cross sectioning. The cell structure is the adult form of medullo-

Fig. 24 (Teng and Katzin). *Case 1881.* A newborn male infant, left eye, nasal side. The large lesion contains examples of each type of rosette.

Fig. 25 (Teng and Katzin). Section through the lesion shown in Figure 24. The retina is replaced, one mm. from the ora, by a fold of nonpigmented epithelium.

epithelioma, and consists of a single layer of columnar or nonpigmented epithelial cells.

2. Rosette formation from irregular remnants of retinoblasts

From embryologic study we know that the picture of the anterior portion of the retina at the 65-mm. stage is very similar to the posterior portion of the retina at the 21-mm. stage. This means that the differentiation of the neuro-epithelium is completed earlier at the posterior portion than the anterior portion where active mitosis or proliferation of retinoblasts lasts longer.

Retinoblast multiplication is supposed to be completed at the sixth month of fetal life, but small remnants of retinoblasts are very commonly found near the ora serrata in the newborn. They are usually in small irregular isolated patches in the retina close to the ora. They may multiply and become

Fig. 26 (Teng and Katzin). High-power section from Figure 25. Note the metaplasia of the folded nonpigmented epithelium adjoining the retina.

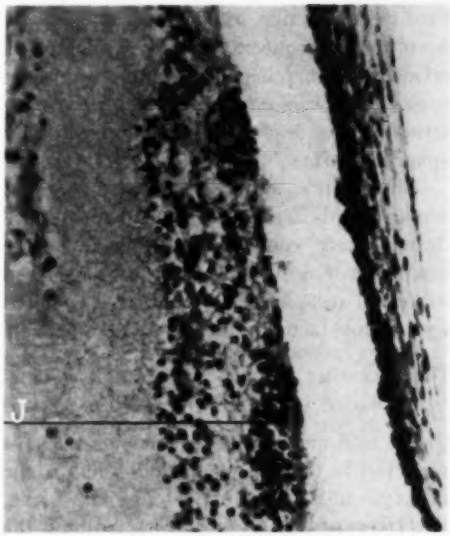


Fig. 27 (Teng and Katzin). High-power section from Figure 25. The external limiting membrane of the retina ends near the bottom of the illustration at (J).

differentiated and often this process is accompanied by rosette formation.

The well-differentiated rosette we classify as Type 1 is similar to the rosette seen in neurocytoma. Type 2 is more like the true rosette of the neuro-epithelioma. Types 3 and 4 are purely proliferated retinoblasts without differentiation and are usually found in the periphery of the retina as distinguished from the better differentiated ones which are more posterior.

We believe that Types 1 and 2 start as proliferations of the remnants of retinoblasts in the external nuclear layer. At certain intervals they differentiate with deposition of a basement membrane, as the retina did in the embryologic stage. Further investigation is needed to classify Types 3 and 4 as cell rests.

3. Rosette formation from retinal folds

We believe the mechanism of rosette and retinal fold formation results from lack of contact of the inner and outer layers of the cup of the retina in the embryonic stage be-

fore the formation of the external limiting membrane. Proliferation of a clump of retinoblasts that begin to elaborate a basement membrane furnishes a supporting structure that leads by surface tension to a spherical form.

The relationship of rosettes to retinal tumors needs more extensive study before drawing any conclusion. We believe that remnants of retinoblasts may be considered to be cell rests and they may be very sensitive to any factor inducing retinal tumor.

II. Phenomena of cytologic and embryologic interest (metaplasia)

The first interesting phenomenon we have observed is that, in congenital folds of the nonpigmented epithelium of the pars plana, the layer of epithelium which adjoins the retina proliferates and differentiates in a manner similar to the cells of the retina, and cells migrate toward the retina to fuse with it.

In early life the nonpigmented epithelial cells have the same potentiality as retinoblasts. In contact with the peripheral retina they may be induced by local growth or other biochemical factors to change into retinal cells. Apparently, cell determination depends upon local environmental factors rather than anatomic concepts of anlage.

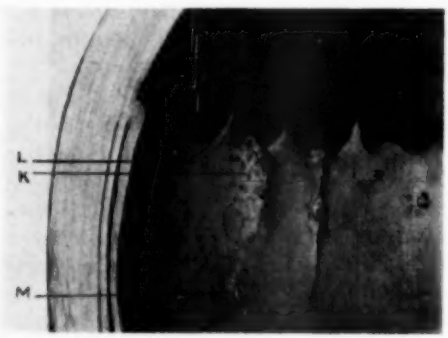


Fig. 28 (Teng and Katzin). *Case 2176.* A 51-year-old man, left eye. Two folds of nonpigmented epithelium and numerous rosettes (Types 1 and 3). (There is only one patch of peripheral cystoid degeneration, so the folds and rosettes do not look granular.)

The second point of cytologic interest is the reversal of cell differentiation. Apparently all cells of neuro-epithelial origin which reach contact with vitreous sooner or later change to the form of nonpigmented epithelium. Folds of nonpigmented epithelium in contact with vitreous retain their original characteristics. Rosettes, however, regardless of type, change their cell characteristics into those of nonpigmented epithelium when they migrate into the vitreous.

In our previous publication concerning vitreous floaters, this point was not appreciated. We presented pathologic granular patch as resulting from proliferation of nonpigmented epithelial cells.

Relationship of rosettes to granular patches

In our first communication of this series we reported that granular patches in the retinal periphery were important in the sequence of development of floater separation and hole formation. The study of babies' eyes has thrown some more light on this process.

By careful examination of eyes of all ages and a stage-to-stage comparison of the pathologic changes, we have come to the conclusion that elongated granular patches form from congenital folds of nonpigmented epithelium of the pars plana. Individual granular patches also develop from the four subtypes of rosettes which we group under Group B (the ones formed from retinoblasts).

Two further cases are presented to illustrate these points, one that of a 51-year-old patient and one of a 60-year-old patient. They show the development of granular patches from epithelial folds and rosettes.

Case 2176

A man, aged 51 years, had his left eye enucleated because of a highly malignant intraorbital melanoblastoma (fig. 28).

The eyeball is normal except for the congenital anomaly in the peripheral retina. There are numerous rosettes over the nasal

side and two small ones on the temporal side of the retina. One Type 1 rosette and at least 40 Type 2 and Type 3 rosettes are scattered within three mm. of the ora. There are two large patches representing nonpigmented epithelial folds.

Histologically, Figure 29 shows the cross section of lesion K, an elongated granular patch of nonpigmented epithelium with many rosettes and a smaller isolated granular patch, a rosette Type 2.

There is senile peripheral cystoid degeneration of the retina, as well as cystoid degeneration of the lesions which make them appear granular. The cells are similar to the nonpigmented epithelium of the pars plana. Note that all nerve cells which migrated inwardly, so that they are in contact with the vitreous, have changed their morphology to resemble nonpigmented epithelium.

In Figure 30, a Type 1 rosette (from M of Figure 28) shows marked cystic degeneration inside the cavity. The cells of the wall retain the morphology of nerve cells because the rosette did not change its position.

Fig. 30 (Teng and Katzin). Section from (M) of Figure 28. Note the Type 1 rosette with degenerative changes.

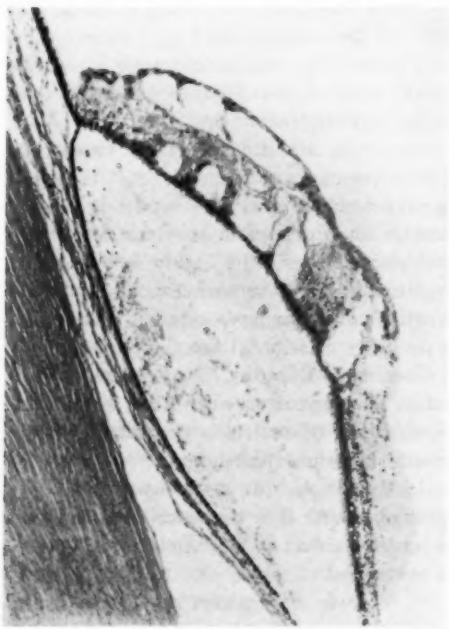


Fig. 29 (Teng and Katzin). Section from (K) of Figure 28. Note the fold of nonpigmented epithelium with cystoid degeneration and adult type medullo-epitheliomalike rosettes. There is peripheral cystoid degeneration external to the fold.

In Figure 31 (L of Figure 28) there are granular patches with cystoid degeneration at their base. There is a marked cystoid degeneration of one of the rosettes. Rosettes in the form of undetached floaters in this older person are comparable to those seen in babies' eyes (figs. 18 and 19).

Case 2234

A man, aged 66 years, died of coronary occlusion. The right eye (fig. 32) is normal except for marked peripheral cystoid degeneration and the congenital defects in the peripheral retina. There are five folds of nonpigmented epithelium in the form of elongated granular patches. Most of them are arranged in the typical way, three in a row with a Type 1 rosette behind the row. The rosette is ring-shaped. The granular

Fig. 31 (Teng and Katzin). Section from (L) of Figure 28. Rosettes are seen in the form of undetached floaters.

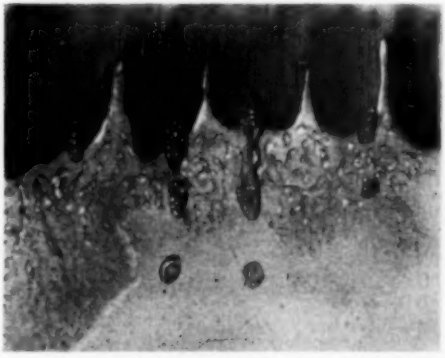


Fig. 32 (Teng and Katzin). *Case 2234.* A 66-year-old man, right eye. Note the gross appearance of folds of nonpigmented epithelium and rosettes (granular patches).

patches are involved by peripheral cystoid degeneration.

In the Type 1 rosette that remained in the retina, there was no migration into the vitreous and it had no formation of vitreous adhesions. The cells are quite comparable to the remainder of the retinal cells.

So, by their incidence, location, arrangement, tendency to formation of local adhesion to the vitreous, cystoid degeneration, floater formation, and cellular character, we can assert that granular patches of the spherical type are the end result of rosettes. These rosettes arise from the remnants of retinoblasts in the periphery of the retina. The elongated granular patches are the end result of the folding of nonpigmented epithelium.

Summary

1. A high incidence of congenital rosettes of the retina was found in babies' eyes. Out of 40 babies, 72 eyes were examined and 17 babies or 23 eyes had rosettes.

2. Three main groups of rosettes were found:

Group A. Rosettes occurring in congenital folds of the nonpigmented epithelium on the pars plana. The cellular character is usually found to be in the form of the adult type medullo-epithelioma.

Group B. Rosette formation from congenital remnants of retinoblasts. This group is very common and it is especially important in the formation of vitreous floaters and retinal holes. The highly differentiated rosettes in this group are very similar to the rosettes found in neurocytomas. Four subtypes were classified from Group B.

Group C. Rosettes in congenital retinal folds. This type is very rare. The appearance of this type of rosette is comparable to the rosette in neuro-epithelioma.

3. Metaplasia of neuro-epithelium has been observed. The presence of potent but opposing factors in the vitreous and retina is postulated.

4. A study of rosettes may serve as an introduction to the histogenesis of retinal tumors.

210 East 64 Street (21).

References

1. Teng, C. C., and Katzin, H. M.: Nonpigmented epithelial cell proliferation and hole formation. Am. J. Ophth., **34**:1237, 1951.
2. Seefelder: Ueber Anomalien im Bereiche des Sehnerven und der Netzhaut Normaler Fotaler Augen. Arch. f. Ophth., **69**:226 and 463, 1909.
3. Lindenfeld, B.: Beitrag zur Bildung Rosetten artiger Figuren in der Netzhaut soust Normaler Fotaler Menschlichen Augen. Klin. Monatsbl. f. Augenh., **51**:443, 1913.
4. Goldstein, I., and Wexler, D.: Rosette formation in the eyes of irradiated human embryos. Arch. Ophth., **5**:591, 1931.
5. Fuchs, A.: Ueber den Anatomischen Befund Eingen Angeborener Anomalien der Netzhaut und des Sehnerven. Arch. f. Ophth., **93**:1, 1917.
6. Jaensch, P. A.: Falten und Rosettenbildungen in der Netzhaut. Arch. f. Ophth., **116**:464, 1925.
7. Joy, H. H., and Delaney, J. H.: From Decoursey and Ash: Atlas of Ophthalmic Pathology, The Army Medical Museum, 1938, pp. 13-14.
8. Gartner, S.: Cyclopia. Arch. Ophth., **37**:220, 1947.
9. Pusey and Brown: Retinal rosette formation of neuroglia in inflammatory processes. Arch. f. Ophth., **32**:325, 1903.
10. Murakami, J.: Ein Beitrag zur Kenntniss der pathologischen Anatomie der Chorioretinitis Disseminata, nebst Bemerkungen ueber das Vorkommen entzuendlichen Rosettenbildungen der Neuroepithelschicht, spontaner Lochbildung in der Macula Lutea und Hyalin-Colloider Kugeln in der Netzhaut. Arch. f. Ophth., **53**:439, 1902.

SUCTION ANTERIOR TO THE IRIS AS A FACTOR IN NARROW-ANGLE GLAUCOMA*

ADOLPH POSNER, M.D.
New York

While it is generally accepted that the rise of tension in narrow-angle glaucoma is associated with a closure of the angle of the anterior chamber, no satisfactory explanation of the relation between these two characteristics has been advanced. Until recently the emphasis was placed on a mechanical crowding and blocking of the filtration angle by the iris; this, in some way, caused by the dilatation of the pupil.

This view resulted from the gonioscopic finding that the angle appeared to be closed during an acute attack. It led to the promulgation of the principle that the treatment should be directed toward pulling the iris root out of the angle with strong miotics, and, if this failed, toward the freeing of at least a part of the angle by means of a broad basal iridectomy.

The widespread employment of gonioscopy has led many clinicians to question the validity of this concept because they frequently found a normal tension in eyes in which the angle remained closed following an iridectomy.

The angle-crowding hypothesis was further shaken by evidence recently presented by Haas and Scheie,[1] as well as by Chandler,[2] that a peripheral iridectomy, which need not be basal, is adequate to relieve the acute attack and to prevent recurrences.

The peripheral iridectomy represents a revival of the peripheral iridotomy which was advocated by Curran[3] some 30 years ago and which, at the time, was favorably received by such authorities as Gifford and Elschnig. The effectiveness, particularly in acute glaucoma, of a poorly executed nonbasal iridectomy was recognized by von Graefe,[4] in 1869, and by Weber,[5] in 1877. Moreover, it is well known that an Elliot trephining operation with a peripheral iridectomy is often effective in acute glau-

* From the Manhattan Eye, Ear, and Throat Hospital. This paper is submitted in recognition of the 70th birthday of Prof. Karl D. Lindner of Vienna.

coma even when no filtering bleb is present. I have under my care several such patients who had trephinations performed for acute glaucoma as long as 20 years ago and who are still maintaining normal tensions. Obviously, the operation, in these cases, functioned as a peripheral iridectomy rather than as a filtering operation.

Haas and Scheie[1] explain the therapeutic action of the peripheral iridectomy the same way that Chandler[2] does. They assume that a relative pupillary block is produced by contact between the iris and lens and that this block permits the hydrostatic pressure in the posterior chamber to become greater than that in the anterior chamber. This increased pressure from behind causes the iris to bulge forward in the shape of a "physiologic iris bombé."

They further point out that a peripheral iridectomy establishes a direct communication between the two chambers; this makes the pupillary block ineffectual and results in a deepening of the entire anterior chamber, accompanied by normalization of tension. Their explanation is similar to the ones proposed by both Curran[3] and Baenziger.[6]

The "pupillary block" hypothesis does not explain how miotics typically produce lowering of the tension, even though contraction of the pupil increases the area of contact between iris and lens and should logically result in an increased pupillary block.

It is true that, if aqueous is to flow from the posterior chamber into the anterior chamber, the pressure behind the iris must be slightly higher than that in front. However, this pressure gradient cannot be significant as a cause of glaucoma; for, if we regard the contact between iris and lens as a valve, the latter should open under a minimal increase of pressure in the posterior chamber.

It has never been proven that relative pupillary block alone can lead to a rise in intraocular pressure. The three isolated observations of Ulbrich, Urbanek and Heine, respectively, which are cited both by Haas and Scheie[1] and by Chandler[2] to prove the existence of a relative pupillary block, are the same as those which Baenziger[6] used in support of his hypothesis.

In each of these three cases, a portion of the iris was congenitally absent and was replaced by a very thin membrane which bulged forward under certain conditions. The original authors in presenting these cases did not claim any relationship to glaucoma, but used them as evidence that the aqueous is formed behind the iris. In not one of these cases was the entire iris pushed forward by the force of the aqueous, nor was any alteration of the intraocular pressure noted.

Both views presumably take for granted that the drainage of aqueous continues until the closure of the angle is complete. However, they underemphasize the role of this same drainage mechanism in creating and subsequently maintaining an obstruction to the outflow of the aqueous.

My concept of the mechanism of narrow-angle glaucoma

It is my opinion that the initiating factor responsible for the rise of tension in narrow-angle glaucoma is the relatively more rapid outflow of aqueous through the corneoscleral trabeculum as compared with the inflow into the chamber angle. In an eye with a shallow anterior chamber or with a narrow angle, the iris may come so close to the cornea, that, in effect, it divides the anterior chamber into two zones: a central zone which maintains normal relationships with the posterior chamber, and a peripheral zone which may be likened to an *annulus* (fig. 1-A).

The two zones are normally in communication with each other, but there are certain conditions which may contribute to bring about actual contact between iris and cornea (fig. 1-B). Among them are dilatation of the pupil, congestion or edema of the ciliary body, and swelling or displacement of

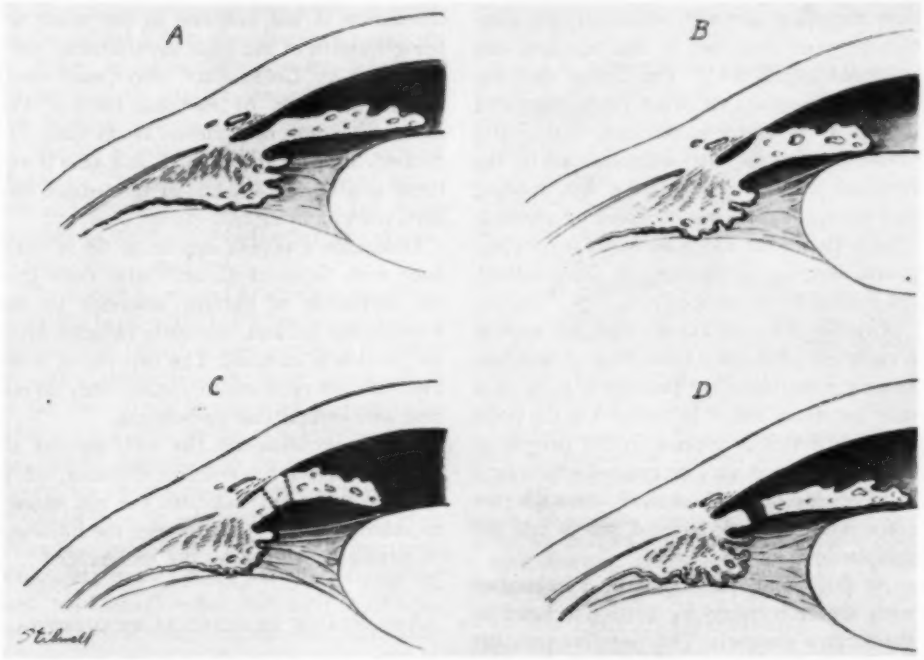


Fig. 1 (Posner). *Development of acute narrow-angle glaucoma and effect of peripheral iridectomy.*
(A) A shallow anterior chamber with narrow angle favors formation of a constriction which divides the chamber into central and peripheral zones.
(B) Dilatation of the pupil leads to contact between iris and cornea and retards the flow of aqueous into the peripheral zone.
(C) The aqueous in the peripheral zone is drained off more rapidly than it can be replaced and the iris bulges forward. The iris is drawn toward the trabeculum and is held there by suction from Schlemm's canal where the hydrostatic pressure is lower than the intraocular pressure. Obstruction of the trabeculum by the iris causes the intraocular pressure to rise.
(D) A peripheral iridectomy establishes a communication between the posterior chamber and the peripheral zone of the anterior chamber. This breaking of the suction relieves the acute attack and prevents recurrences.

the lens. This contact, while it interferes with free circulation of aqueous, does not necessarily result in complete obstruction.

The drainage of aqueous into the canal of Schlemm takes place within the peripheral zone of the anterior chamber. It has been established by many observers that in the normal eye, outflow of aqueous is relatively constant at a rate of approximately 2.0 cu. mm. per minute.

Goldmann[7] has shown that the hydrostatic pressure in Schlemm's canal and in the aqueous veins is normally about 10 mm. Hg. If we assume that the normal intraocular pressure is 20 mm. Hg, then we have a pressure differential of 10 mm. Hg between the anterior chamber and the canal of Schlemm.

Goldmann states that this differential or "outflow pressure" is used up in overcoming the resistance of the corneoscleral trabeculum. This applies under conditions of unimpeded circulation.

When the peripheral zone or *annulus* is cut off from the normal reservoir of aqueous, a somewhat different pressure pattern is found. Aqueous continues to drain from the peripheral zone until exhausted, at which

time the space becomes obliterated by contact between the root of the iris and the trabeculum (fig. 1-C). The reason that the aqueous continues to drain until exhausted is that it is under a constant hydrostatic pressure (20 mm. Hg) accompanied by the forward displacement of the iris moving into the space formerly occupied by aqueous. This is the factor which accounts for the apparent bulging of the iris or the so-called, "physiologic iris bombé."

This sequence of events does not require a complete blockage of the flow of aqueous from the central to the peripheral zone. It is only necessary that it be reduced to the point where a deficit is created in the peripheral zone. The deficit may be created either by a relative rapidity of outflow through the trabeculum or by a reduced inflow into the peripheral zone.

At this point, the iris is held in contact with the trabeculum by suction created in the outflow channels. This negative pressure (10 mm. Hg) equals the difference between the pressure in Schlemm's canal (10 mm. Hg) and the normal intraocular pressure (20 mm. Hg).

The interference with normal outflow causes the intraocular pressure to rise. The elevation of pressure is not wholly accounted for by secretion. The hypertonic nature of the aqueous causes it to absorb water. This increases the volume of the aqueous and, since the outflow channels are obstructed, the intraocular pressure rises. Kinsey[8] estimates that, in the absence of any outflow, the hypertonicity alone can increase the intraocular pressure to as much as 200 mm. Hg.

As a result of higher intraocular pressure, aqueous may be forced past the obstruction into the peripheral zone. If sufficient aqueous is so forced in, it may separate the iris root from the trabeculum. In this way the suction is broken, the angle opens, and the tension returns to normal. Clinically, this is described as a spontaneous termination of an acute attack.

The view that the obstruction to aqueous circulation is not absolute in the sense of being watertight has been satisfactorily substantiated by Goldmann,[9] who found that, under conditions of elevated tension, the rate of outflow of aqueous is, in fact, increased to twice the normal, and that it returns to a normal level after the tension has been reduced by an iridectomy.

Goldmann's results appear to be at variance with those of Grant[10] who finds that the coefficient of outflow, obtained by the tonographic method, is greatly reduced when the tension is elevated. The two views, however, are not contradictory, since they do not deal with comparable phenomena.

Goldmann measures the total amount of aqueous leaving the anterior chamber, while Grant's coefficient indicates the net change in volume of the eyeball under the influence of pressure exerted by the tonometer.*

Application to clinical phenomena

Clinically, the noncongestive phase is the first phase of acute narrow-angle glaucoma. It is characterized by an elevation of tension, a variable amount of corneal edema, and by the absence of congestion, pain, or marked loss of vision. The noncongestive phase may subside either spontaneously or under the influence of miotics.

Spontaneous remissions are frequent in the early stages of glaucoma. The mechanism of the remission is not clear. It is possi-

---

* Grant's coefficient of outflow denotes the volume change per minute in cu. mm. for each mm. Hg increase in intraocular pressure. Goldmann (Abflussdruck, Minuten-volumen and Widerstand der Kammerwasserströmung des Menschen, Documenta Ophthalmologica, 5-6: 278-356, 1951) showed that Grant's coefficient of outflow approximates the reciprocal of Goldmann's coefficient of resistance, since the latter also is a function of both outflow pressure and minute volume. In acute glaucoma, Goldmann found the minute volume increased, but not as much as would be expected from the high intraocular pressure. He, therefore, assumes that the resistance is also increased. Since the resistance is increased, its reciprocal, the coefficient of outflow, is necessarily diminished.

ble that compression of the vorticose veins by the raised intraocular pressure results in a higher intravascular pressure, which is transmitted by way of the orbital veins to the anterior scleral plexus of veins, thus raising the pressure in the aqueous drainage channels. This retards the outflow of aqueous and provides an opportunity for more aqueous to accumulate in the peripheral zone of the anterior chamber.

The action of miotic drugs in acute glaucoma has been subjected to various interpretations. The miotic action probably depends on a straightening of the bend in the plane of the iris which would tend to break the suction (negative pressure) which holds the iris root in apposition to the trabeculum. As a corollary to this, the response to the miotic would be directly proportional to the degree of angulation of the iris. It has been my clinical experience that eyes with a relatively deep anterior chamber but with a narrow angle show a more consistently favorable response to miotics than do eyes with a shallow chamber and a uniformly vaulted iris (fig. 2).

The noncongestive phase passes into the congestive phase when the interference with the circulation exceeds a certain threshold. If the initial rise of tension is brought about by a forward displacement of a congested ciliary body, the noncongestive phase of the glaucoma may be greatly shortened in duration.

When the iris remains pressed against the wall of the eyeball for a considerable time, the blood vessels in the iris are compressed. This often results in an atrophy of the iris, either total or sector-shaped, a characteristic sequel to acute glaucoma. If the iris atrophy involves the entire thickness of the iris, a "window" results which can serve the same purpose as a peripheral iridectomy.

This explanation of the pathogenesis of narrow-angle glaucoma is suggested as a working hypothesis since it interprets, better than any previous view, the clinical pattern of the disease.

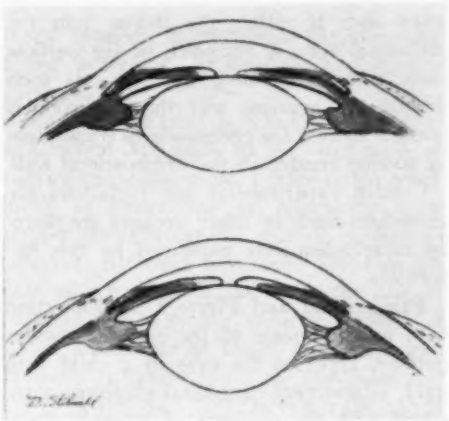


Fig. 2 (Posner). To illustrate the relation of the depth of the anterior chamber to the efficacy of miotics on the iris suction mechanism in narrow-angle glaucoma. In a narrow-angle eye the same amount of pupillary miosis produces more traction on the iris root when the anterior chamber is deep (upper drawing) than when it is shallow (lower drawing).

Related observations

In a narrow-angle eye, when the anterior chamber is opened by means of a scratch incision made at the limbus, only a minute quantity of aqueous escapes through the incision since the iris is pressed into the wound and seals it off. In several instances, I noticed that, even after I had introduced a spatula into the incision, the anterior chamber did not collapse as long as the spatula remained in the peripheral zone. In four eyes, the tension was measured with the Schiøtz tonometer before the incision was made and, again, after the aqueous was permitted to escape from the peripheral zone. The volume lost was estimated by referring to Friedenwald's table (See Moses and Bruno[11]).

All four eyes were operated upon in the noncongestive stage. The average amount of aqueous evacuated from the peripheral zone was 5.0 cu. mm. ($\pm$1.0). If we assume that the average volume of the anterior chamber was 100 cu. mm., the peripheral zone contained only five percent of the total volume. From this, it is easily seen how, in the

intact eye, if either the inflow into the peripheral zone is retarded, or the outflow from it is accelerated, this space is soon drained of aqueous and the iris root is pressed against the corneoscleral trabeculum by suction created in the corneoscleral wall.

Certain experimental and fortuitous observations made by other workers are thereby more adequately explained by this hypothesis.

Friedenwald and Pierce,[12] in connection with measurements of the rate of aqueous outflow, reported the case of a child with mild, intermittent congenital glaucoma associated with the presence of a small, dislocated lens in the anterior chamber. They observed that the tension rose whenever the lens came to lie over the pupil so as to form a ball valve with the iris. At first the iris bulged slowly forward around the edge of the lens. Within one-half hour, the anterior chamber became empty. "It was not until the iris had approached close to the cornea that the intraocular pressure began to rise."

In the light of the present hypothesis, it is obvious that the pupillary block permitted the aqueous to be drained out of the anterior chamber. A "physiologic iris bombé" was formed, converting the deep chamber into a shallow one. After the aqueous contained in the anterior chamber had been evacuated, the iris root itself was drawn up against the trabeculum and the intraocular pressure began to rise.

Similar hydrodynamic factors probably are responsible for the glaucoma which may follow the introduction of air into the anterior chamber. According to Barkan,[13] air glaucoma will occur only if the air bubble produces a seclusion of the pupil. The following is quoted from Barkan's excellent description of a clinical case.

"In the first case (wide-angle glaucoma with vision reduced to counting fingers) it was observed five hours after operation that the iris had crawled up the posterior surface of the lower third of the cornea in the shape of a localized iris bombé and blocked the corresponding portion of the angle. The remaining chamber was filled with air. That portion of the angle which was not blocked by iris was evidently blocked by air, judging from the marked increase in tension recorded."

As in the case described by Friedenwald and Pierce, the glaucoma in this case and in similar cases of seclusion of the pupil can be easily explained by the "suction hypothesis." This is further corroborated by Barkan's own observation that "in cases in which the anterior chamber was completely filled with air, it was found necessary to maintain postoperative tension below 20 mm. Hg (McLean) in order to avoid a secondary rise." This tension is approximately equivalent to 15 mm. Hg (Schiøtz). At this pressure level, there is an approximate hydrodynamic equilibrium between Schlemm's canal and the anterior chamber so that the outflow does not exceed the inflow.

These observations support the "suction hypothesis" by demonstrating that a relative block at any point between the posterior chamber and the corneoscleral trabeculum may initiate an acute rise in ocular tension; a shallow chamber and a narrow angle are not absolute prerequisites, but merely predisposing factors. The essential requirement for the initiation of ocular hypertension is that, in the peripheral zone, the rate of outflow must exceed the rate of inflow.

Therapeutic considerations

The practical value of the "suction hypothesis" is based on its applicability to therapy, both pharmacologic and surgical. This phase will be but briefly discussed, and only to the extent to which it bears on the thesis of this paper.

The classical treatment of acute narrow-angle glaucoma both in the noncongestive and the congestive stages consists in the instillation of miotic drugs. As previously noted, the miotics act by drawing the iris away from the corneoscleral trabeculum, and breaking the suction. If the suction is broken

at any point of the circumference, the effect on the tension and on the depth of the chamber is the same as if the iris were drawn away along its entire circumference.

In the noncongestive phase, it is best to begin treatment with a weak miotic such as pilocarpine. If the tension still remains elevated after maximal miosis of the pupil has been attained, it may be better to increase the frequency and the strength of dosage than to resort immediately to stronger miotics, particularly DFP. Little additional mechanical effect, if any, can be expected from the stronger miotic, whereas its powerful action on the blood vessels and muscles of the ciliary body is apt to precipitate the congestive phase.

With regard to surgical measures, the "suction hypothesis" supplies an explanation for the effectiveness of a peripheral iridectomy or even an iridotomy. A seclusion of the peripheral *annulus* from the main body of aqueous is rendered impossible so long as a communication, no matter how small, is present between the posterior chamber and the periphery of the anterior chamber. The iridectomy, in effect, transforms the narrow-angle eye into a wide-angle eye by making available to the outflow channels the entire reservoir of aqueous contained in the posterior chamber (fig. 1-D).

A peripheral iridectomy is superior to a complete iridectomy, both from the cosmetic and the optical points of view. When a fistulizing operation is desired, the peripheral tab of iris is left attached at its base and brought through the incision. This results in a "peripheral iridencleisis" which is usually as effective as the classical iridencleisis.

The safest incision in narrow-angle glaucoma is an ab-externo incision made either at the limbus or one-half millimeter behind it. In a case in which the tension is normal between attacks, an iridectomy is the operation of choice. An iridencleisis should not be performed routinely, but only when definitely indicated. It is more likely to be complicated by delayed reformation of the anterior chamber, formation of peripheral anterior synechias, or possibly a malignant glaucoma.

The "iris suction" hypothesis may lead to a different approach to the treatment of narrow-angle glaucoma. I refer to the possibility of preventing or alleviating acute attacks by reducing the rate of outflow of aqueous. Attempts have been made in the past with partial success, as in the case of "glaucosan," a strong vasoconstrictor, the use of which was advocated by Hamburger. However, a rationale for the treatment was not presented and the criteria for the use of the drugs were inadequately worked out. The clinical application of the "iris suction" hypothesis to the treatment of narrow-angle glaucoma is in the experimental stage.

Summary

While crowding of the chamber angle by the iris and pressure behind the iris are both factors in acute narrow-angle glaucoma, the primary cause of closure of the angle is suction exerted anterior to the iris because of the relatively lower pressure in Schlemm's canal.

Shallowness of the anterior chamber favors formation of a constriction which divides the chamber into central and peripheral zones. By this constriction, the flow of aqueous into the peripheral zone is so retarded that it does not keep pace with the outflow through the canal of Schlemm. As aqueous in the peripheral zone is drained off, the iris bulges forward in the form of an iris bombé to fill the space previously occupied by the aqueous.

During this phase, the intraocular pressure remains normal. Since the hydrostatic pressure in Schlemm's canal is lower than the intraocular pressure, the formation of the iris bombé and the tendency toward closure of the angle can be regarded as a result of suction in front of the iris rather than of pressure from behind.

After the peripheral zone is drained of aqueous, the iris presses against the trabecu-

lum and obstructs the outflow; this causes the intraocular pressure to rise.

An analogous mechanism is observed in glaucoma resulting from the presence in the anterior chamber of a dislocated lens or of air, or from other factors causing seclusion of the pupil.

A spontaneous remission may occur when either the outflow is retarded or the inflow into the peripheral zone is accelerated.

Miotics act in narrow-angle glaucoma by breaking the suction which holds the iris in contact with the trabeculum. Once the suction is broken, the entire anterior chamber deepens and the angle opens.

The surgical treatment consists in forming a communication between the posterior chamber and the peripheral zone of the anterior chamber; this, in effect, converts a narrow-angle eye into a wide-angle eye by making available the reservoir of aqueous contained in the posterior chamber. This may be accomplished either by a complete or a peripheral iridectomy, or even by an iridotomy. A peripheral iridectomy is considered preferable to a complete iridectomy for cosmetic and optical reasons.

By applying this "iris suction" hypothesis, it may be possible to develop a method of influencing the rate of outflow of aqueous in order either to alleviate or prevent an acute attack.

667 Madison Avenue (21).

References

1. Haas, J. S. and Scheie, H. G.: Peripheral iridectomy in narrow-angle glaucoma. Tr. Am. Acad. Ophth., **56**:589 (July-Aug.) 1952.
2. Chandler, P. A.: Narrow-angle glaucoma. Arch. Ophth. **47**:695 (June) 1952.
3. Curran, E. J.: New operation for glaucoma involving a new principle in the etiology and treatment of chronic primary glaucoma. Arch. Ophth., **49**:131, 1920.
4. v. Graefe, A.: Beitraege zur Pathologie und Therapie des Glaukoms. Arch. f. Ophth., **15**:108, 1869.
5. Weber, A.: Die Ursache des Glaukoms. Arch. f. Ophth., **23**:1, 1877.
6. Baenziger, T.: Die Mechanik des acuten Glaukoms und die Deutung der Iridektomiewirkung bei demselben. Ber. ü. d. Versammlung d. deutsch. Ophth. Gesellsch., **43**:43, 1922.
7. Goldmann, H.: An analysis of primary glaucoma. Tr. Ophth. Soc. U. Kingdom, **69**:455-476, 1949.
8. Kinsey, V. E.: A unified concept of aqueous humor dynamics and the maintenance of intraocular pressure. Arch. Ophth., **44**:215 (Aug.) 1950.
9. Goldmann, H.: Abflussdruck, Minuten-volumen und Widerstand der Kammerströmung des Menschen. Documenta Ophth., **5-6**:278, 1951.
10. Grant, W. M.: Clinical measurements of aqueous outflow. Arch. Ophth., **46**:113 (Aug.) 1951.
11. Moses, R. A., and Bruno, M.: The rate of outflow of fluid from the eye under increased pressure. Am. J. Ophth., **33**:389, (Mar.) 1950.
12. Friedenwald, J. S. and Pierce, H. F.: Circulation of the aqueous. I: Rate of flow. Arch. Ophth., **7**:538 (Apr.) 1932.
13. Barkan, O.: Glaucoma induced by air blockade. Am. J. Ophth., **34**:567 (Apr.) 1951.

Ophthalmic Miniature

From this remarkable history, it seems manifest, that the dilatation of the pupil soon after the coming on of the coma, was owing to the compression of the thalamic nervorum opticorum by the water collected in the brain which rendered the retina insensible to the stimulus of light.

Robert Whytt, *Essay on the vital and other involuntary motions of animals,* Edinburgh, 1751.

(Presented the first analysis of the pupillary light reflex, called for some time thereafter "Whytt's reflex.")

COMPARATIVE EFFECTS OF CORTISONE ON RABBIT EYES TREATED BY CYCLODIATHERMY AND CYCLO-ELECTROLYSIS*

L. BENJAMIN SHEPPARD, M.D.
Richmond, Virginia

Numerous reports have appeared in the literature demonstrating the beneficial effects of cortisone and ACTH in inflammatory ocular diseases,[1-6] since Hench and others[7] established its usefulness in 1948 as a therapeutic agent. Cortisone has proved especially effective in the treatment of diseases of the anterior segment of the globe.[8, 9]

The exact mechanism of the action of cortisone is not known. In ocular tissue, the hypersensitivity reaction to inflammatory or to toxic substances is favorably suppressed by its administration.[1, 2, 5, 10] The authors point out that cortisone inhibits inflammatory cellular infiltration, hyperemia, and edema associated with reduced capillary permeability. However, no such beneficial effect is exerted upon the progress of an associated underlying infectious process. Similarly, there is a suppression of the normal mesenchymal tissue response consequent upon surgical procedures, with retardation of the healing process.[11-15]

The purpose of this paper is to report briefly the effects of the use of cortisone on rabbit eyes, following treatments by cyclo-electrolysis and by cyclodiathermy. The technique used has been previously explained.[16]

I. EXPERIMENTAL PROCEDURE

Five mongrel rabbits, aged approximately four to six months, in which there was no evidence of eye infection, were selected. Two rabbits were used as the controls, while the other three were treated with cortisone. One control rabbit was killed at the end of five days, the other after one month. The cortisone-treated animals were killed at the end of five days, one month, and two months, the last having been treated again at the end of the first month. Control animals were treated with diathermy and electrolysis, using the method previously described.[16]

CONTROL ANIMALS

Rabbit 46 (Control)—no cortisone used. Weight, 2,700 gm.; aged four to six months.

RIGHT EYE. (Day 1) Chemosis, hyperemia at root of iris with some fibrinous exudate; (Day 2) iridocyclitis increased with corneal clouding; (Day 3) essentially unchanged; (Day 4) essentially unchanged; (Day 5) keratitis mild.

LEFT EYE. (Day 1) Slight clouding of the cornea adjacent to treated area, moderate chemosis; (Day 2) iridocyclitis mild, corneal clouding unchanged; (Day 3) chemosis decreased; corneal clouding unchanged; (Day 4) same; (Day 5) unchanged.

The rabbit was killed and the eyes were enucleated.

Rabbit 72 (control)—no cortisone used. White albino; weight, 2,500 gm.; aged four to six months.

Treatment. Cyclodiathermy, O.D., and cyclo-electrolysis, O.S. Killed at the end of one month.

RIGHT EYE. (1) Early changes, first week: Marked hyperemia and chemosis with slate-gray zone in treated area; iris slate-gray adjacent to treated area; moderate iridocyclitis. (2) Intermediate changes, second week: Marked chemosis and hyperemia; beginning vascularization of periphery of the cornea. (3) Late changes, third and fourth week: Normal and clear.

LEFT EYE. (1) Early changes, first week: Same as O.D., but less marked. (2) Intermediate changes, second week: Slight chemo-

* From the Department of Ophthalmology, Medical College of Virginia. This work was supported by a grant from the Snyder Ophthalmic Foundation. Acknowledgment is made to Merck and Co., Inc., Rahway, New Jersey, for a supply of cortisone, and to the Medical College of Virginia for the use of laboratory facilities.

sis; very slight hyperemia. (3) Late changes, third and fourth week: Normal and clear.

The rabbit was killed and the eyes were enucleated.

TREATED ANIMALS RECEIVING CORTISONE

1. Cyclodiathermy was done to the right eye and cyclo-electrolysis to the left eye; 30 punctures were made in the superior temporal third of the globe, two rows parallel to and equidistant from the limbus at one and two mm. posterior to the limbus between the superior and the lateral recti muscles.

2. The eyes were treated with cortisone injections on alternate days—four days preoperative, the day of operation, and four days postoperative. One cc. of cortisone suspension was injected subconjunctivally. Thereafter cortisone ointment was applied daily.

3. Dosage of cortisone. "Cortisone Merck" containing 25 mg. per cc. was mixed with three parts normal saline, a 1:4 dilution with each cc. containing 6.25 mg. of cortisone. One cc. of this solution, or 6.25 mg., was injected subconjunctivally.

4. Atropine was applied daily.

Five-day experiment

Rabbit 75 (cortisone-treated). White albino, aged four to six months; weight, 2,500 gm.

RIGHT EYE. (Day 1) Gray-white cortisone plaque under conjunctiva in treated area, mild chemosis in vicinity of treated area; (Day 2) subconjunctival cortisone plaque, moderate chemosis, thinning of sclera in treated area, cornea cloudy adjacent to treated zone; (Day 3) exudation superior one-third of globe with marked chemosis, gross hemorrhage into angle of anterior chamber with organized plastic exudate; (Day 4) increase in inflammatory reaction around site of punctures, clouding of cornea over superior half with increased iridocyclitis, iris retracted at puncture site, possibly herniation of iris and ciliary body through ruptured sclera in treated area; (Day 5) same as Day 4 except that severe iridocyclitis and keratitis progressed inferiorly.

LEFT EYE. (Day 1) Mild chemosis over treated area; (Day 2) very slight chemosis at treated area, otherwise normal in appearance, conjunctival cortisone plaque; (Day 3) normal in appearance except slight hyperemia over treated zone; (Day 4) unchanged, plaques of cortisone still present; (Day 5) slight chemosis over treated zone with slate-gray areas in sclera at puncture sites.

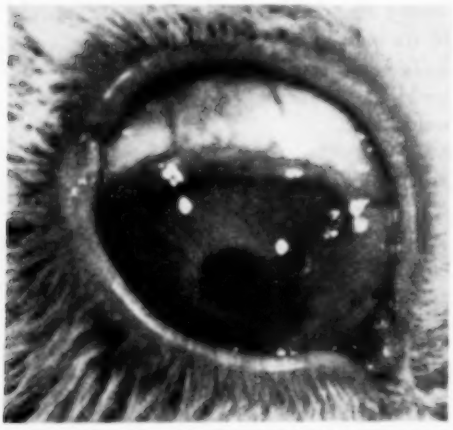


Fig. 1-A (Sheppard). *Rabbit 72 (control).* Right eye (cyclodiathermy) on fifth postoperative day, showing moderate chemosis and hyperemia.

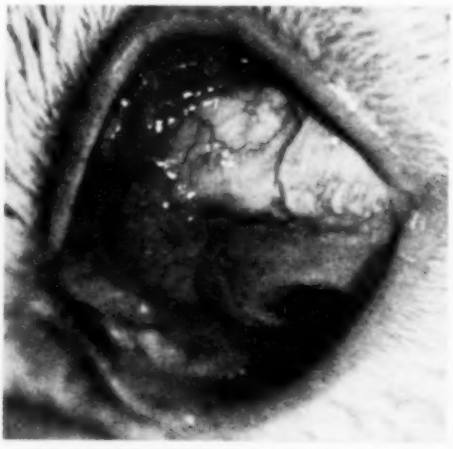


Fig. 1-B (Sheppard). *Rabbit 72 (control).* Left eye (cyclo-electrolysis) on fifth postoperative day, showing the reaction to be essentially the same.

Fig. 2-A (Sheppard). *Rabbit 72 (control).* Right eye (cyclodiathermy) at the end of the first postoperative month, showing hyperemia and slate-gray areas in treated zone of sclera. (Note Figure 10, a microphotograph.)

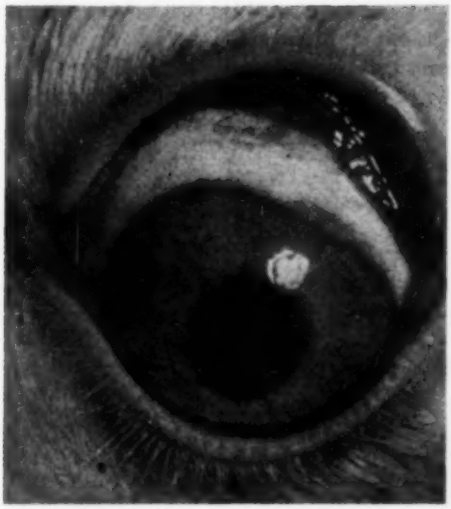
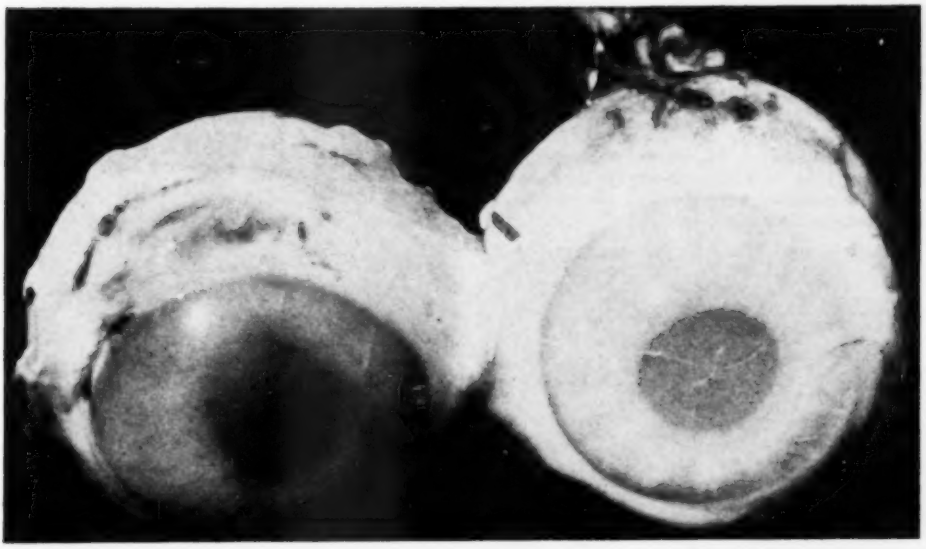


Fig. 2-B (Sheppard). *Rabbit 72 (control).* Left eye (cyclo-electrolysis), showing essentially the same reaction as in Figure 2-A but less marked. (Note Figure 11, a microphotograph.)



A B

Fig. 3 (Sheppard). *Rabbit 75 (cortisone-treated)* killed on the fifth postoperative day. (A) Right eye (cyclodiathermy), showing rupture of the sclera in the treated zone, severe iridocyclitis, clouding of the superior half of the cornea, hemorrhage, and organized plastic exudate extending through the pupil into the anterior chamber. (Note Figure 8, a microphotograph.) (B) Left eye (cyclo-electrolysis), showing very slight chemosis over treated zone and slate-gray areas in sclera. (Note Figure 9, a microphotograph.)

The animal was killed and both eyes were enucleated. Figure 3 shows the fresh specimen.

One-month experiment

Rabbit 74 (treatment with cortisone). White albino, aged four to six months; weight, 2,200 gm.

RIGHT EYE. (Day 1) Moderate chemosis, mild iridocyclitis and white cortisone plaque at treatment site, questionable elevation of intraocular pressure; (Day 2) Moderate chemosis, plaque still present; (Day 4) inflammation increasing, slate-gray iris adjacent to treated area with sclera white over area, hyperemic conjunctiva, moderate iridocyclitis, and hypotension; (Day 5) unchanged, note Figure 4-A; (Day 7) marked slate-gray iris, marked iridocyclitis, suppuration over treated area, and hyperemia of conjunctiva; (Day 9) mild keratitis, moderate hyperemia, purulent conjunctivitis, slight chemosis over treated area, slate-gray area of iris with sclerosed, necrotic, thrombosed vessels, and intense hyperemia in remainder of iris, no cortisone plaques observed; (Day 13) marked hyperemia, purulent conjunctivitis, moderate chemosis, peripheral ulcer superior one-third of cornea with early pannus, keratitis over remainder of cornea, contracted pupil and hyperemia of iris; (Day 14) perforation at limbus adjacent to treated area under conjunctiva, marked chemosis, eye soft, necrotic conjunctiva and tenacious exudate, corneal findings as for Day 14 (fig. 4-C); (Day 18) corneal ulceration extending; (Day 26) completely ulcerated cornea, panophthalmitis; (Day 30) atrophic, shrunken globe with a solid cheeselike material extending from globe.

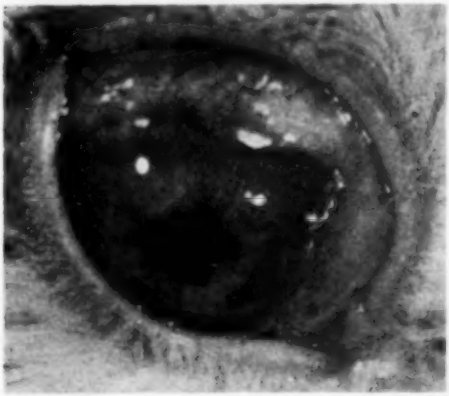


Fig. 4-A (Sheppard). *Rabbit 74 (cortisone-treated).* Killed after one month. Right eye (cyclodiathermy) on the fifth postoperative day, showing moderate chemosis and hyperemia over the treated zone and moderate iridocyclitis.

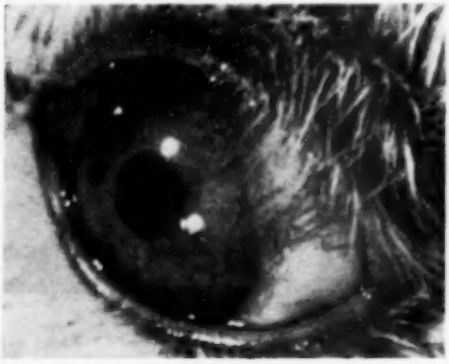


Fig. 4-B (Sheppard). *Rabbit 74.* Left eye (cycloelectrolysis), fifth postoperative day, showing slight chemosis over the treated zone.

Fig. 4-C (Sheppard). *Rabbit 74.* Same as Figure 4-A but at the end of the second postoperative week, showing marked chemosis, tenacious conjunctival exudate, rupture of globe at limbus in treated zone. A panophthalmitis followed, progressing to atrophy of the globe. (Note Figure 12, a microphotograph.) The left eye became normal at the end of the second postoperative week and was still normal at the end of one month. (Note Figure 13, a microphotograph.)

LEFT EYE. (Day 1) Moderate chemosis over treated zone, white cortisone plaque, iridocyclitis mild; (Day 2) slight chemosis with plaque still present; (Day 4) slight chemosis; (Day 5) unchanged, note Figure 4-B; (Day 7) mild chemosis over treated area; (Day 9) essentially normal, no cortisone plaques observed; (Day 13) clear and normal; (Day 14) normal; (Day 18) normal; (Day 26) normal; (Day 30) normal.

The rabbit was killed and the eyes were enucleated.

Sixty-day experiment

Rabbit 81 (treated with cortisone). Eyes treated again after 30 days with same procedure. The rabbit was killed at the end of 60 days. White albino, aged four to six months; weight, 2,200 gm.

On the first day *both eyes* appeared to have suffered little, as evidenced by mild hyperemia at the treated sites. Cornea and sclera were normal with no clouding noted.

RIGHT EYE. (Day 2) Slight chemosis and hyperemia, conjunctival plaques from cortisone injections; (Day 3) mild chemosis and hyperemia; (Day 4) moderate chemosis and hyperemia, moderate hyperemia of iris with a two-mm. slate-gray area at the treated site, subnormal tension; (Day 7) mild hyperemia and chemosis; (Day 9) clear and normal, no objective changes for the remainder of the month; (Day 30) clear and normal. treated with cortisone and cyclodiathermy punctures, as originally; (Day 35) mild reaction for first four days after treatment, marked chemosis and hypermia, white cortisone plaques at injection site; (Day 39) slight chemosis, otherwise clear; (Day 46) clear and normal with slate-gray discoloration of sclera at treated site; (Day 60) clear and normal.

LEFT EYE. (Day 2) Same as O.D.; (Day 3) same as O.D.; (Day 4) mild hyperemia; (Day 7) same as O.D.; (Day 9) same as O.D.; (Day 30) clear and normal, treated with cortisone and cyclo-electrolysis punctures, as originally; (Day 35) slight reaction with white cortisone plaques; (Day 39) same as O.D.; (Day 46) same as O.D. except less discoloration; (Day 60) same as O.D.

The rabbit was killed and the eyes were enucleated.

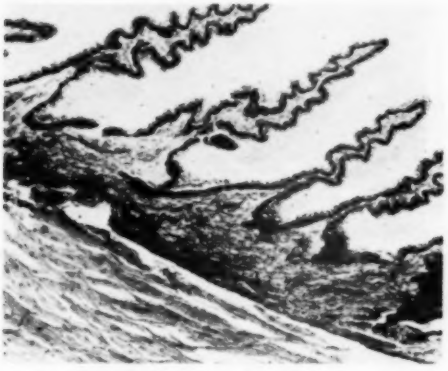


Fig. 5-A (Sheppard). *Normal ciliary body and ciliary process,* showing sclera, spaces of Fontana, ciliary body, and ciliary processes.

Fig. 5-B (Sheppard). High-power view of normal ciliary process, showing stroma of process, middle zone, pigmented epithelium, and nonpigmented epithelium, outer layer.

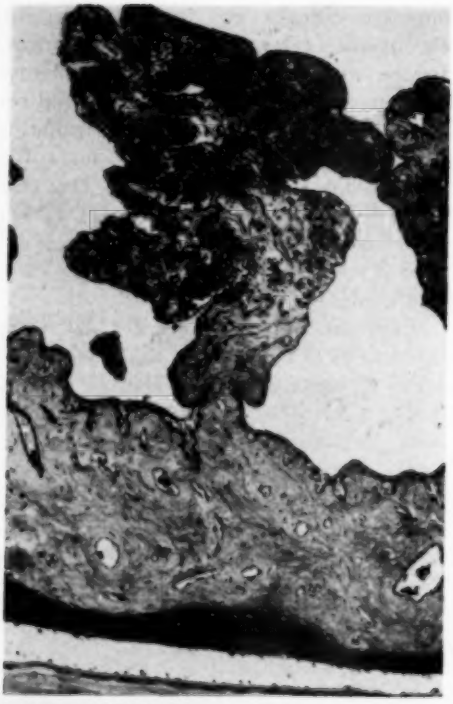


Fig. 6-A (Sheppard). *Rabbit 46 (control)* on the fifth postoperative day. Right eye (cyclodiathermy), showing edema, hyperemia, and cellular infiltration of ciliary body and ciliary processes. Note subciliary fluid.

Fig. 6-B (Sheppard). A high-power view of Figure 6-A, showing edema, cellular infiltration, engorgement of the vessels, and hemorrhage in ciliary processes.

II. Results

The postoperative course was uneventful in the eyes of the control animals compared with other animals similarly treated (figs. 1 and 2). In the cortisone-treated animals, the right eyes of Rabbit 74 and Rabbit 75 (diathermy-treated) showed little difference from the control in the first two days, but

Fig. 7-A (Sheppard). *Rabbit 46 (control).* Fifth postoperative day. Left eye (cyclo-electrolysis), showing edema, cellular infiltration of ciliary body, and ciliary processes. Note subciliary fluid.

thereafter they showed markedly increased reaction, with little or no response of the treated tissue to repair and poor resistance to a purulent infection.

This infection invaded the right eye in Rabbit 75 on the third day, leading to softening of the sclera and rupture of the globe. This also occurred in the right eye of Rabbit 74, though it started at a later date (seventh postoperative day). The left eye (electrolysis-treated) had a much milder postoperative course (figs. 3 and 4).

Rabbit 81, treated in the same manner and

Fig. 7-B (Sheppard). High-power view of Figure 7-A, showing ciliary process with edema, cellular infiltration, engorgement of vessels, and hemorrhage.

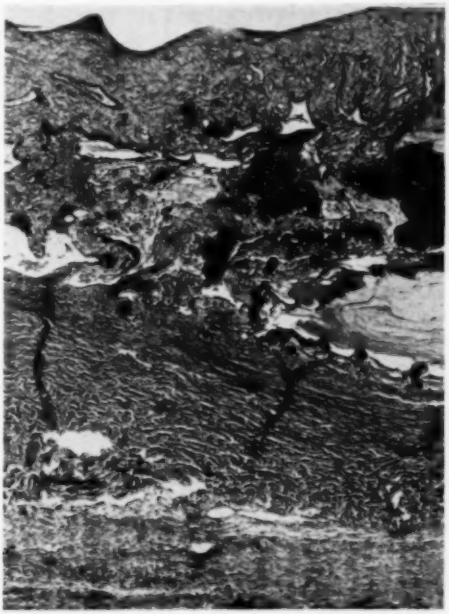


Fig. 8 (Sheppard). *Rabbit 75 (cortisone-treated).* Fifth postoperative day. Right eye (cyclodiathermy), showing hemorrhage in ciliary body, cellular exudate, and edema to be less than in the five-day control. The cellular reaction is mostly lymphocytic.

Fig. 9 (Sheppard). *Rabbit 75 (cortisone-treated).* Left eye (cyclo-electrolysis), showing slight cellular infiltration, lack of vascularity, and edema. Note difference between this and the five-day control in Figures 7-A and 7-B.

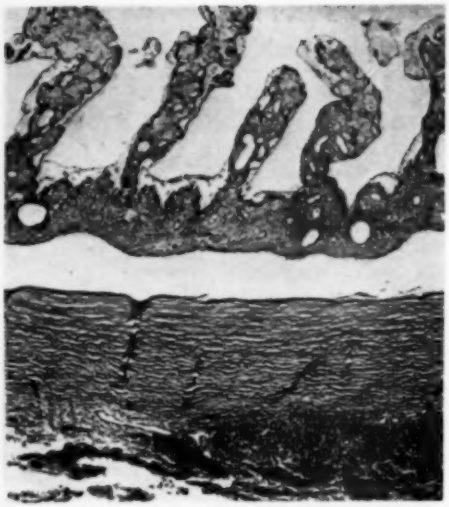


Fig. 10-A (Sheppard). *Rabbit 72 (control).* End of first postoperative month. Right eye (cyclodiathermy), showing cellular infiltration of conjunctiva and episclera, edema of ciliary processes, and engorged vessels.

Fig. 10-B (Sheppard). High-power view of Figure 10-A, showing ciliary process. Note edema, capillary proliferation, and engorged vessels.

treated again after 30 days, showed little variation from the control. The eye tissues did not become infected.

III. Histopathologic findings

Frontal sections were made on all eyes. Note Figure 5, normal eye.

The histopathologic changes in the rabbit eyes show some important effects of cortisone. The eyes of the cortisone-treated rabbits showed much less edema and cellular inflammatory response in both early and later stages. Comparison of the controls (figs. 6, 7, 10, and 11) with the cortisone-treated eyes (figs. 8, 9, 12, 13, and 14) demonstrates the differences in reaction of the conjunctiva, sclera, ciliary body, and ciliary processes in the control and in the cortisone-treated animals.

In the cortisone-treated animals there was a suppression of the mesenchymal response to treatment that was observed in the controls. The reduction in the initial fibrin response and subsequent fibroblastic prolifera-

Fig. 11 (Sheppard). *Rabbit 72 (control).* Left eye (cyclo-electrolysis), showing a ciliary process with eosinophilic cellular response, edema, increased vascularity, and loss of nonpigmented epitnelium.

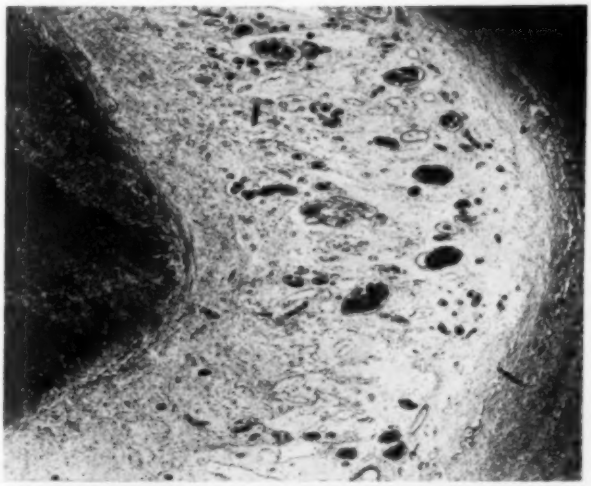


Fig. 12-A (Sheppard). *Rabbit 74 (cortisone-treated)* at the end of first postoperative month. Right eye (cyclodiathermy), showing moderate edema, cellular infiltration of the sclera and ciliary body, chiefly by lymphocytes. There is secondary infection of the globe with abscess formation and subsequent rupture. (Abscess—left third; ciliary body—middle third; sclera—right third.) Compare this picture with Figures 10-A and 10-B.

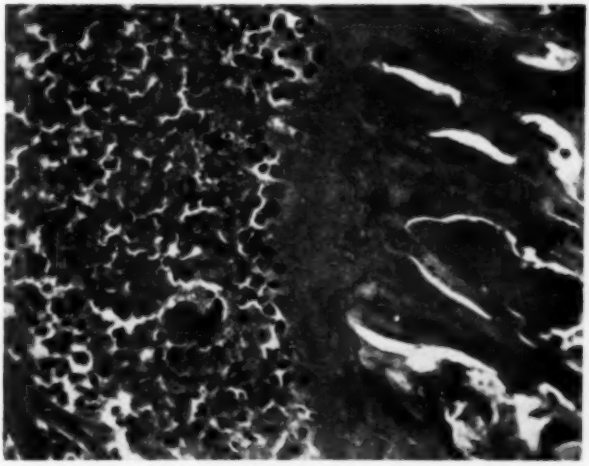


Fig. 12-B (Sheppard). High-power view of Figure 12-A, showing, at junction of abscess and ciliary processes, cells in abscess and edematous ciliary processes.

Fig. 13 (Sheppard). *Rabbit 74 (cortisone-treated).* End of first postoperative month. Left eye (cyclo-electrolysis), showing no edema and less vascularity than control. No fibroblastic activity. Compare ciliary processes with those shown in Figure 11.

Fig. 14 (Sheppard). *Rabbit 81 (cortisone-treated).* Right eye (cyclodiathermy), treatment repeated at the end of 30 days. The eye was enucleated at the end of the second postoperative month. This picture shows that there is no edema, small hemorrhage at the tip of the process, less cellular response and edema than is shown in the one-month control. The left eye, treated with cyclo-electrolysis, showed even less change.

tion has been well established.[11, 12] In the right eyes of Rabbit 74 and Rabbit 75, there was an ectasia at the site of treatment. The control eyes withstood infection better than those that were cortisone-treated. This illustrated experimentally a finding well known clinically, that the patient may succumb to an infection without showing signs of toxicity. Cortisone masks the signs of infection while the infection spreads (figs. 8 and 12).

There are less vascularity and fibroblastic response in the cortisone-treated animals. (Compare Figure 7 with Figure 9 and Figure 11 with Figure 13.) This demonstrates that cyclodiathermy is comparatively a more destructive process than cyclo-electrolysis and that in regeneration the normal connective-tissue response is reduced by cortisone.

IV. Conclusions

From the observation of this limited series of cortisone-treated eyes, it appears that the clinical advantages gained by having an eye comfortable and with reduced inflammation after surgery are striking. On the other hand, cortisone may mask a serious infection or retard the normal processes of healing where strength of adhesion is most desired. It seems advisable, therefore, not to use cortisone immediately after ocular surgery, or, if its use is necessary, to administer antibiotics at the same time.

Medical Arts Building.

I wish to express my appreciation to Dr. Gordon Hennigar, Department of Pathology, Medical College of Virginia, for his kind assistance and consultation, and to thank Mrs. Florence Herold, Mr. Charles J. Townsend, and Mr. Earl Fox for their laboratory assistance. I am deeply grateful to Mr. Neal Grubb for his active interest.

References

1. Woods, A. C.: ACTH and cortisone in ocular disease. Am. J. Ophth., **33**:1325, 1950.
2. Beigel, A. C.: Effect of cortisone on horse serum uveitis in rabbits. Arch. Ophth., **45**:258, 1951.
3. Olson, J. A., et al.: Use of ACTH and cortisone in ocular disease. Arch. Ophth., **45**:274, 1951.
4. Scheie, H. G., et al.: ACTH and cortisone in ophthalmology: Report of cases. Arch. Ophth., **45**:301, 1951.
5. Mosher, H. A.: Local use of cortisone in ophthalmic diseases. Arch. Ophth., **45**:317, 1951.
6. Steffensen, E. H., et al.: Topical cortisone in treatment of anterior segment eye disease. Am. J. Ophth., **34**:345, 1951.
7. Hench, P. S., et al.: Cortisone and ACTH in clinical medicine. Proc. Staff Meet. Mayo Clinic, **25**:474, 1950.
8. Agatston, H.: The use of cortisone in ocular conditions. Am. J. Ophth., **34**:1655-64, 1951.
9. Gordon, Dan M., et al.: The use of ACTH and cortisone in ophthalmology. Am. J. Ophth., **34**: 1675-86, 1951.
10. Kendall, E. C., et al.: A physiologic and chemical investigation of the suprarenal cortex. Proc. Am. Soc. Biol. Chem., J. Biol. Chem., **116**: LVII, 1936.

11. Dunnington, J. S.: The effect of sutures and of thrombin upon ocular wound healing. Tr. Am. Acad. Ophth., Nov.-Dec., 1951.
12. Newell, F. W., and Dixon, J.: Effect of subconjunctival cortisone upon the immediate union of experimental corneal grafts. Am. J. Ophth., **34**:977, 1951.
13. Baker, B. L., and Whitaker, W. L.: Interference with wound healing by local action of adrenocortical steroids. Endocrinology, **46**:544, 1950.
14. Ragan, C., et al.: Effect of cortisone on production of granulation tissue in the rabbit. Proc. Soc. Exper. Biol. & Med., **72**:718, 1949.
15. Creditor, M. C., et al.: Effect of ACTH on wound healing in humans. Proc. Soc. Exper. Biol. & Med., **74**:245, 1950.
16. Berens, C., Sheppard, L. B., and Duell, A. B.: Cyclo-electrolysis for glaucoma. Am. J. Ophth., **34**:53-70, 1951.

NONPERFORATING CYCLODIATHERMY IN THE TREATMENT OF GLAUCOMA*

DIATHERMIA RETROCILIARIS

A. URRETS-ZAVALÍA, JR., M.D., AND A. URRETS-ZAVALÍA, M.D.
Cordoba, Argentina

In the last few years, it has been usual to consider antiglaucomatous operations as divided into those intended to create a new and artificial way of drainage for the aqueous—external and internal fistulizing operations—and those devised to inhibit, in a certain measure, the formation of aqueous at the anterior segment of the uvea by means of the cauterization of the ciliary body (cyclodiathermy).

Such a separation is not justified because the first-mentioned interventions do not act principally on the base of the fistulas they create, nor do the latter retard, in the ordinary sense, the formation of the aqueous (Weekers,[50] François,[14,15] Duke-Elder[11]).

Curran,[8] in 1925, and Fiore,[12] in 1930, succeeded in reducing abnormally elevated ocular pressure by the transclerotic galvanocauterization of the ciliary body.

Weve,[57,58] in 1932, found that frequently the pressure remains normal, for some time at least, in eyes submitted to a superficial extensive anterior diathermization of the sclera because of anterior retinal disinsertions. He was able to reduce ocular tension in some cases of infantile glaucoma by means of a superficial diathermization at the ciliary zone.

Amsler,[2] later obtained similar results. Nevertheless, it was Vogt[37-43] who, since 1936, employed systematically multiple diathermic micropunctures of the ciliary body (Zyklodiathermiestichelungen), in order to decrease ocular tension in most of the different forms of glaucoma.

The indications for cyclodiathermy, confined at the beginning to cases with very bad prognosis, in which it was impossible to try another operation with any probability of success, were progressively extended.

Vogt,[42] in one of his later publications on the subject, could affirm that this intervention was then ready to compete with the so-called fistulizing operations in the treatment of primary chronic glaucoma.

Subsequently, Franceschetti,[13] Wagner,[44-47] Wagner and Richner,[49] Richner,[26] Wagner, Karbacher, Meyer, Wolf, and Weber,[48] Gasteiger,[16] Meesmann,[20] Stocker,[28] Benner,[5] Marr,[19] Meyer,[21] and Sautter[27] have employed Vogt's operation in its original form, but with different results.

It is now possible to compare the bad results announced recently by Marr[19] and the mediocre ones by Meyer,[21] with the conclusions of Benner[5] and Sautter.[27] According to these authors, cyclodiathermy must be considered the operation of choice in most cases

* Presented at the Sociedad de Oftalmologia de Cordoba, May 25, 1951.

of secondary glaucoma, in those of advanced primary glaucoma with severe restrictions of the visual field, and in those of primary glaucoma already operated upon with another procedure; without success.

In their opinion, preference should be given to fistulizing interventions in most cases of chronic simple or congestive glaucoma, and to iridectomy in cases of acute glaucoma. We believe with Stratton[29] that, for the most part, the untoward results obtained are due to the fact that cyclodiathermy was generally confined to cases of absolute or hemorrhagic glaucoma, and seldom employed in favorable cases which are usually operated upon with other methods, the prestige of which is in this way increased.

Vogt's primary operation has undergone many modifications during recent years. Some of them (Thiel's[31] subscleral cyclodiathermy, Redslob's[24] perforating transconjunctival cyclodiathermy, Moreu's[22] goniocyclodiathermy, and Díaz-Domínguez's[10] diathermic cyclodialysis) have not found general acceptance.

In another group of antiglaucomatous operations, such as those proposed by Guerry,[17] Arato,[3] and Ketessy,[18] not very well known as yet, diathermy is used with the object of decreasing the afflux of blood to the ciliary body, so as to diminish the formation of the aqueous by destroying the ciliary arteries; hence the name of angiodiathermy by which these procedures have been designated.

Other techniques, on the contrary, which have met with increasing approval, are developments of the method suggested by Weve's first experiments, already mentioned. In them, the application of high-frequency currents is superficial. Thus, Albaugh and Dunphy,[1] in 1942, obtained good results in 25 out of 32 patients by cauterizing the previously denudated sclera with a flat electrode four mm. from the limbus. Subsequently, Villaseca,[36] with a similar procedure, obtained only mediocre results.

L. and R. Weekers,[31] in 1942, devised their method of nonperforating, transconjunctival cyclodiathermy, characterized by the peculiarities suggested by its name and by the fact that the cauterizations are performed at seven or eight mm. from the limbus.

According to them, the hypotensive effect of the operation is due to the massive uveal vasodilation to which diathermy gives rise, and to the subsequent modifications of the rate of formation and resorption of the intraocular fluid. Later on, they announced their first results which were successful, if we take into account the fact that they refer to cases with bad prognosis, and described their technique which comprises the use of Coppez's pyrometric—or rather thermometric—electrode. They also pointed out the innocuousness of the procedure, studied the mechanism of its hypotensive action, and determined its clinical indications.[32-36]

It would seem from the experience gathered by these and other authors that the unquestionable efficacy of the method depends less on the fact that the applications are nonperforating and transconjunctival and are performed simultaneously around the whole perimeter of the globe, or on the possibility of establishing exactly the thermal action by means of a special electrode, than on applying the current not at the level of the pars plana but at the posterior limit of the ciliary body or even behind the ora serrata.

That is why Arruga,[4] who wisely coined the name of *diathermia retrociliaris*, performs the operation without dissecting the conjunctiva with a common perforating or half-perforating electrode.

Reiser[25] applies a flat electrode directly at the two inferior quadrants of the previously denudated sclera. Verrey[34] and Desvignes and Naudin[9] use an electrode with a flat extremity, two mm. in diameter, consequently larger than that of Weekers.

We do not have a thermometric electrode so we employ a common flat one 0.75 mm. in diameter, similar in this respect to that of Weekers. In spite of these differences, these

authors' results and ours are practically the same.

We believe that this new type of cyclodiathermy represents the most outstanding contribution to glaucoma surgery in the last 10 years. This is due, of course, to the good results obtained, comparable, however, to those recently reported by Sautter,[27] using Vogt's technique, as well as to the fact that this procedure does not have such serious complications as are sometimes observed with original cyclodiathermy—severe iridociliary reactions with formation of synechias, severe intraocular infections, opacities of the lens, marked hypotony followed by phthisis bulbi, pronounced anesthesia or hypoesthesia of the cornea sometimes accompanied by epithelial erosions or even by ulcerations, neuroparalytic keratitis, hypopyon keratitis, scleral ulcerations, vitreous loss, retinal hemorrhages, retinal detachment, and even, in some cases, sympathetic ophthalmia (Vogt,[39, 41, 43] Streiff,[30] Thiel,[31] Palomar,[23] Díaz-Domínguez[10]).

Although our observations have not been carried on for as long a time as we would desire, we wish to communicate our present excellent results with Weekers' technique because we consider the procedure without equal in many cases and because it is still relatively little known in America.

Technique

As far as possible we have followed the indications established clinically and experimentally by L. and R. Weekers.[51–54]

The patient should be well sedated (Verrey[35]). A five-percent cocaine solution is instilled repeatedly in the conjunctival fornices. A retrobulbar injection of 1.5 to 2.0 cc., of two-percent novocaine-adrenalin is made. In certain cases, 20-percent alcohol is added to the novocaine-adrenalin solution.

After anesthesia, which has to be carefully executed, is complete, a suture is placed through each rectus muscle so the eye can always be put in the desired position; otherwise the operation is sometimes difficult to perform (Verrey[34]).

Fig. 1 (Urrets-Zavalía). The nonperforating electrode.

A series of 12 to 20 diathermy applications of 15 seconds each is placed round the whole circumference of the globe, seven to nine mm. from the limbus by means of a special electrode devised for that purpose (fig. 1). Muscular insertions are usually avoided so that the anterior ciliary vessels will not be damaged (Arruga[4]). The use of this number of cauterizations and their application to the whole perimeter of the eye arises from the desire to affect immediately the whole ciliary body.

In spite of the fact that the temperature reached on the surface is the same when a current of high intensity is used during a short time, as when a current of lower intensity is used for a longer period, the reaction in the depth of the tissues is greater with the lower current (Coppez[7]). Accordingly, the intensity of the current must be graduated so that application is relatively prolonged.

Since the applications are made seven to nine mm. from the limbus—that is, at the level or a little behind the ora serrata—the complications observed with Vogt's original procedure, which are much more frequent as the punctures approach the limbus, are avoided.

Vogt[42] advised not to come closer than 2.5 mm. to the cornea in order to act on the pars plana of the ciliary body but not on the corona ciliaris. L. and R. Weekers[53] prefer to operate at a distance of seven to nine mm. from the limbus, basing their decision on the fact that the intense uveal vasodilation which then takes place, and which for them is the essential reaction to diathermic treatment, extends only a little backward to the choroid; whereas, owing to local vascular

and nervous peculiarities, it extends forward to the whole ciliary body and the iris.

In another publication (Urrets-Zavalía, Jr.[33]) the mechanism of action of cyclodiathermy has been discussed. We believe its hypotensive effect depends not on uveal vasodilation, which, unless it were exclusively or nearly exclusively capillary (and it is not), constitutes in itself a hypertensive factor, but on the fact that, as a result of diathermic application, the secretory activity of the ciliary epithelium ceases and the aqueous is no longer hypertonic as compared to the blood plasma.

We agree with L. and R. Weekers[51] that, with nonperforating electrodes, a good hypotensive effect is obtained, an effect not inferior to that produced with perforating ones. Perforations are not only unnecessary but harmful because they may give rise to greater complications. Although we have sometimes employed Lacarrere's electrode, we definitely prefer the one we are now using, which, like that of Weekers,[52] has a blunt and flat end, 0.75 mm. in diameter, and is coated with an insulating enamel.

Insulation is an important point because otherwise, when the electrode is pressed as close to the uvea as possible, current would be lost through the conjunctiva which is flattened by the end of the electrode but remains in contact with its lateral surface. When the electrode is pressed against the globe, we can see at the points of application, even before turning on the current, the slatelike color of the choroid through the conjunctiva and the sclera, thinned by the compression.

Although we do not have a thermometric electrode, we have become able through experience to graduate exactly the diathermic action. A row of minute burns of the conjunctiva and the sclera is produced. These heal in a couple of days.

Since the electrode is so small in size, we are able to treat a very limited area, far from the ciliary processes, from the iris, the lens, and the cornea. In addition, it is not necessary to dissect the conjunctiva for the current produces only minute foci of necrosis on this tissue.

We do not think that Coppez's electrode is any more essential in this procedure than it is in the operation for retinal detachment. It may even give only apparent precision, since it measures the temperature at the level of the conjunctival surface, not taking into account the thickness of the underlying episcleral and scleral tissues which may be quite variable.

The intensity of the current, measured by the thermic milliamperimeter with which the apparatus is provided, was generally about 60 ma. in our first experiments and about 50 ma. in later ones. These figures are, for many reasons, only approximate. We have always acted with extreme caution, guided by the principle that it is preferable to have to repeat the operation than to harm the eye with excessive current.

We have now succeeded in establishing the minimum intensity capable of satisfactorily reducing the pathologically elevated ocular tension without giving rise to unfavorable reactions. Accordingly, our results have improved day by day.

Verrey[35] advises irrigation of the eye with cold saline solution between the applications, so as to prevent overheating the globe which, in his opinion, is responsible for the transitory hypertension which sometimes follows the operation.

Saline irrigation is, indeed, a useful measure. However, when the pressure rises to a marked degree, it is advisable to perform—as Arruga[4] does—one or two diathermic transconjunctival punctures, 10 or 12 mm. from the limbus, with a more intense current, so as to give passage to a pearl of vitreous. This is particularly wise in cases of narrow-angle, congestive glaucoma. We have never observed such excessive rises in tension as those pointed out by Reiser,[25] which are due, we believe, to an exaggerated diathermic action.

We have always measured the ocular ten-

sion at the beginning of the operation, 10 minutes after the retrobulbar injection is given, and again at the conclusion. The fall in tension which follows the injection is frequently a clue to the degree the pressure will drop in response to diathermic treatment. The hypotensive effect is noticeable at the moment of the first dressing 24 hours later, and it becomes more marked in the subsequent days. It is possible, generally, to unbandage the eye 48 hours after operation.

If the intraocular pressure should again rise and cannot be reduced by miotics, the intervention may be repeated one or several times.

Material and Results

At first we performed this operation only on patients who could not undergo any other without extreme risk—secondary glaucoma of a varied nature, absolute or hemorrhagic glaucoma, chronic glaucoma with very restricted visual field and poor visual acuity, and congenital glaucoma. Later, encouraged by the results obtained, we resorted to this operation in cases of less doubtful prognosis.

To facilitate the summary of our observations, our cases are divided into four groups: (1) Secondary glaucoma; (2) chronic simple or congestive glaucoma of protracted evolution and unfavorable prognosis; (3) chronic glaucoma of a less serious prognosis; and (4) congenital, absolute, and anterior glaucoma.

Changes of the visual field mentioned herein always refer to the central isopters, investigated with the smallest discernible white test objects, generally 1/2,000 or 2/2,000.

1. Secondary glaucoma (Chart 1)

Case 1

B. F., aged 28 years, was seen on November 17, 1950, with glaucoma of the left eye secondary to severe contusion of the globe which had been followed by profuse hemorrhage into the anterior chamber. The hemorrhage was already absorbed, but marked atrophy of the iris and opacity of the lens were present. Tension was 51 mm. Hg (Schiøtz). Vision was perception of light.

On November 25, 1950, diathermia retrociliaris was performed on the left eye. The tension pre- and postoperative was 44 mm. Hg. On November 27th, conjunctival congestion and pronounced chemosis did not permit the tension to be measured. No iridociliary reaction was present.

On December 2, 1950, the left eye showed slight congestion and chemosis and the tension was 18 mm. Hg. By January 13, 1951, there was no congestion and the tension was 25 mm. Hg.

A second diathermia retrociliaris was performed on the left eye on April 16, 1951 (arrow in Chart 1, Curve 1). The preoperative tension was 31 mm. Hg; the postoperative, 24 mm. Hg. By April 17th, the left eye showed only slight chemosis and tension was 15 mm. Hg. On July 31st, there was no conjunctival reaction and the tension was 19 mm. Hg.

If the intraocular pressure remains normal, cataract extraction will eventually be attempted.

Case 2

D. A., aged 63 years, was seen on July 6, 1950. The aphakic right eye showed pronounced upward displacement of the pupil and extreme clouding of the vitreous. A 24-hour tension curve showed the base and peak pressures to be 25 and 35 mm. Hg. (Schiøtz). Vision was counting fingers at 30 cm.

Since it was considered necessary to attempt substitution of the vitreous at a later date, diathermia retrociliaris was performed on October 12, 1950. The preoperative tension was 18 mm. Hg; postoperative, 25 mm. Hg. On October 13th the right eye showed slight chemosis and no iridociliary reaction; tension could not be measured. By October 19th there was no chemosis and the pressure was 10 mm. Hg. On December 13th, tension in the right eye was 13 mm. Hg; vision was counting fingers at 30 cm.

Vitreous substitution was done on December 20, 1950 (see arrow on Chart 1, Curve 2), according to the technique developed by one of us (Urrets-Zavalia, Jr.[33]). By April 23, 1951, the fundus showed a red reflex and its details were well visualized. There appeared to be no macular lesions; the disc was of normal color; tension was 10 mm. Hg; vision was 1/25, corrected.

Case 3

G. S., aged 55 years, was seen on October 20, 1950, with glaucoma of the right eye secondary to violent contusion of the eyeball, with posterior luxation of the lens. There was slight edema of the cornea; tension was 44 mm. Hg (Schiøtz); vision was 20/70 with a +11.0D. lens.

Diathermia retrociliaris was performed on the right eye on October 21, 1950. Preoperative tension was 52 mm. Hg; postoperative, 60 mm. Hg. On October 23rd, the right eye showed pronounced chemosis but neither corneal edema nor an iridociliary reaction. Tension was 37 mm. Hg. By the next day, October 24th, tension was 21 mm. Hg; the chemosis persisted.

On May 7, 1951, the right eye showed no con-

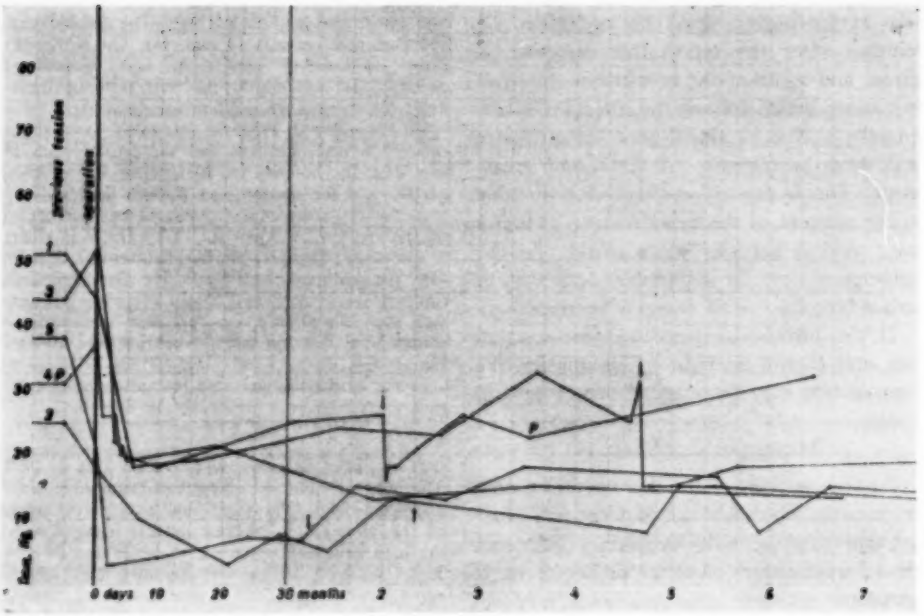


Chart 1 (Urrets-Zavalía). SECONDARY GLAUCOMA. Each individual curve corresponds to the tensional variations of one of the observations referred to in the text. (P) Tension under pilocarpine.

junctival reaction; tension was 15 mm. Hg; vision with a +12.0D. lens was 20/20.

CASE 4

H. A., aged 39 years, was seen on January 29, 1949, with glaucoma of the right eye secondary to penetration of a foreign body which pierced the iris obliquely, passed through the lens without opacifying it, and could not be located ophthalmoscopically, biomicroscopically, nor radiographically.

There was moderate excavation of the disc; tension was 50 mm. Hg (Schiøtz), and uncorrected vision was 20/25. There was marked narrowing of the visual field.

Since medical treatment was unsuccessful, a trephining operation was performed on February 22nd, and by March 10th the right eye showed a good filtering scar; tension was 18 mm. Hg; vision was 20/60, uncorrected.

When seen on November 22, 1949, the scar in the right eye was completely flattened and seemed to be obliterated. The excavation of the disc was more pronounced than before. Tension was 30 mm. Hg, and uncorrected vision was 20/30. The visual field had become even more constricted.

One-percent pilocarpine was ordered three times a day. By February 21, 1951, the condition of the right eye was unchanged and tension was 31 mm. Hg under pilocarpine. Uncorrected vision was 20/25 and the visual field was still more reduced.

Diathermia retrociliaris was performed on the right eye on February 23rd. Preoperative tension was 37 mm. Hg; postoperative, 31 mm. Hg. The next day the eye showed slight chemosis and the tension was 26 mm. Hg. By March 5th, no chemosis was present and tension was 18 mm. Hg. Uncorrected vision was 20/40. By April 23rd, tension was 26 mm. Hg and uncorrected vision, 20/25. The visual field had suffered further narrowing.

A second diathermia retrociliaris was performed on the right eye on April 24, 1951 (see arrow on Chart 1, Curve 4). Preoperative tension was 26 mm. Hg; postoperative 8.0 mm. Hg. On April 26th, there was slight conjunctival congestion and tension was 18 mm. Hg. By May 18th, the right eye showed no reaction and the tension was 26 mm. Hg, with uncorrected vision of 20/25. Two-percent pilocarpine twice a day was ordered. On September 6, 1951, tension in the right eye was 32 mm. Hg, uncorrected vision was 20/20, and the visual field was unchanged.

CASE 5

V. A., aged 25 years, was seen on September 22, 1949, with glaucoma of the left eye secondary to a full-blown sympathetic ophthalmia. There were numerous keratic deposits, a large number of floating cells in the anterior chamber, multiple posterior synechias, and almost complete pupillary occlusion. Tension was 38 mm. Hg (Schiøtz) and vision was hand movements at 50 cm. The right eye had been enucleated.

Under treatment, the tension became slightly but

not permanently reduced. The inflammatory symptoms disappeared, however.

Before attempting an optic iridectomy, it was decided to perform diathermia retrociliaris on September 27, 1950. The preoperative tension was 21 mm. Hg; postoperative, 8.0 mm. Hg. Atropine was used. On September 28th, the left eye showed great chemosis, a clear cornea, and tension was 13 mm. Hg. By October 7th, there was very slight chemosis but there were two small marginal ulcers of the cornea at the 3- and 9-o'clock positions. No iridociliary reaction was present and the tension was 4.0 mm. Hg. Occlusion, atropine, and topical penicillin were prescribed. By October 21st, the corneal ulcers had healed. Tension was 4.0 mm. Hg.

On November 10th, a superior iridectomy was performed (see arrow, Chart 1, Curve 5) which eventually became obliterated in spite of local cortisone therapy. By August 27, 1951, the left eye showed no inflammatory symptoms, tension was 14 mm. Hg, and vision was counting fingers at one meter.

COMMENT

In four of these five cases of secondary glaucoma, diathermia retrociliaris succeeded in normalizing the ocular tension, sometimes after a transient phase of marked hypotony. In the fifth case (Case 4), that due to perforating injury of the globe, after an unsuccessful fistulizing operation, diathermy produced an insufficient drop in tension, in spite of being performed twice within an interval of two months; nor could miotics keep the tension within normal values in this case.

During the first postoperative days, chemosis in varying degrees was always noted. Although in one case (Case 5) there were two corneal marginal ulcers, no uveal reaction was ever detected.

2. CHRONIC SIMPLE OR CONGESTIVE GLAUCOMA OF PROTRACTED COURSE AND UNFAVORABLE PROGNOSIS (Chart 2)

CASE 1

C. M., aged 53 years, was seen on August 28, 1947, with a wide-angle, chronic simple glaucoma of the left eye. There were excavation and pallor of the disc, with marked narrowing of the visual field. Tension under pilocarpine was 33 mm. Hg (Schiøtz). Corrected vision was 20/60.

On November 3, 1950, diathermia retrociliaris

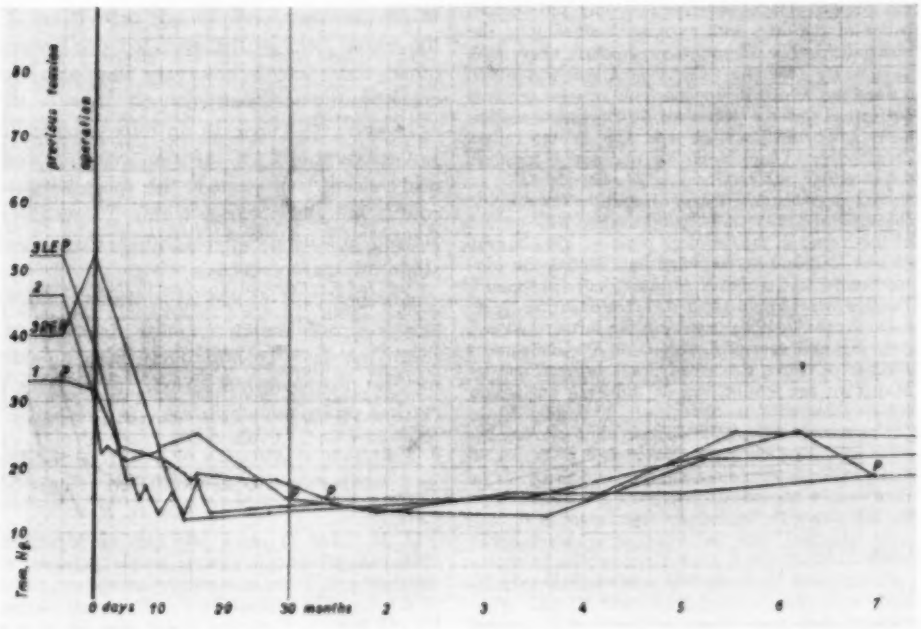


Chart 2 (Urrets-Zavalia). CHRONIC SIMPLE OR CONGESTIVE GLAUCOMA OF PROTRACTED COURSE AND UNFAVORABLE PROGNOSIS. Each individual curve corresponds to the tensional variation of one of the observations referred to in the text. (P) Tension under pilocarpine.

was performed on the left eye. The preoperative tension was 31 mm. Hg, which was also the postoperative reading. On November 4th, although there was no iridociliary reaction, severe chemosis prevented taking the tension. On November 6th, the chemosis of the left eye still persisted, the pupil was round, large, and reacted only slightly to light.

The chemosis remained unchanged on November 8th; there were numerous floating cells in the anterior chamber. Several small posterior pupillary synechias were present. The tension was 18 mm. Hg, and corrected vision was 1/20. A one-percent homatropine ointment was applied.

On the next day, November 9th, the condition of the left eye remained unchanged with the exception that the cornea showed increased hypesthesia as well as a half-moon-shaped marginal ulcer. Tension was 18 mm. Hg. One-percent homatropine drops were prescribed and occlusion ordered.

By November 17th, the corneal symptoms and the iridociliary reaction had subsided, although some of the pupillary synechias were still present. Tension was 13 mm. Hg and vision 1/20, corrected.

When examined on August 20, 1951, the left eye showed no reaction; tension was 18 mm. Hg and corrected vision was 20/50. There were no further alterations in the field.

CASE 2

P. P., aged 53 years, was seen on November 3, 1949, with wide-angle, chronic simple glaucoma of the left eye. There were deep cupping and marked pallor of the disc, with extreme narrowing of the visual field. The 24-hour base-pressure curve was 43 mm. Hg (Schiøtz). Corrected vision was 20/60.

On October 25, 1950, diathermia retrociliaris was performed on the left eye. Preoperative tension under pilocarpine was 30 mm. Hg; postoperative, 25 mm. Hg. The left eye showed slight chemosis the next day and tension was 22 mm. Hg.

By November 4th, the left eye still showed some chemosis and there was a light marginal clouding of the cornea at the 9-o'clock position. Tension was 22 mm. Hg and corrected vision, 20/100. On November 8th, the corneal infiltration had disappeared. Tension was 12 mm. Hg and corrected vision 20/70.

On April 30, 1951, tension in the left eye was 25 mm. Hg and corrected vision, 20/70. One-percent pilocarpine, three times daily, was ordered and by May 23rd the tension was 18 mm. Hg and vision could be corrected to 20/60.

The visual field of the operated left eye has become somewhat narrower, although it is similar to that of the glaucomatous, but unoperated, right eye. However, a normal tension level is maintained in the left eye with the aid of miotics.

CASE 3

S. A., aged 55 years, was seen on February 16, 1950, with chronic congestive, narrow-angle glaucoma of both eyes. There was marked excavation of the discs. Vision was: R.E., 20/50, corrected; L.E., light perception. The visual field of the right eye was extremely narrow. Tension with pilocarpine was: R.E., 40 mm. Hg (Schiøtz); L.E., 52 mm. Hg.

A diathermia retrociliaris was performed on the left eye on October 10, 1950, and one on the right eye on October 25th. With the exception of marked chemosis in both eyes, the postoperative course was uneventful. One-percent pilocarpine, once a day, was ordered for the right eye on October 21st; for the left eye on November 7th.

When seen on June 7, 1951, tension was: R.E., 21 mm. Hg; L.E., 24 mm. Hg; vision was: R.E., 20/30, corrected; L.E., light perception. The visual field of the right eye was unchanged. In all other respects the condition of both eyes was unchanged.

COMMENT

In the four eyes in this group, two with chronic simple glaucoma and two with chronic congestive glaucoma, the onset of the disease had been insidious and the prognosis was unfavorable.

After medical treatment had failed, cyclodiathermy with a current of 50 to 60 ma. normalized the ocular tension either alone or with the aid of miotics.

Visual acuity which was lower in the immediate postoperative weeks later returned to its previous level. In one case (Case 2) the visual field in the operated eye became more constricted; however, this also occurred in the nonoperated eye.

In one case (Case 1) an intense iridociliary reaction and an inferior marginal corneal ulcer complicated the postoperative course but disappeared within a few days, leaving no after effects other than some slight pupillary synechias.

Another case (Case 2) suffered a slight marginal infiltration of the corneal parenchyma near the horizontal meridian during the postoperative period. In all cases a marked, transient chemosis was present.

3. CHRONIC GLAUCOMA OF SHORTER COURSE AND MORE FAVORABLE PROGNOSIS (Chart 3)

CASE 1

D. M., aged 44 years, was seen on March 30, 1951, with wide-angle chronic simple glaucoma of the right eye. There was excavation without pallor of the disc. The 24-hour base pressure was 44 mm. Hg (Schiøtz). Corrected vision was 20/60 and there was moderate narrowing of the visual field. Pilocarpine (one percent, six times a day) was ordered for the right eye. On April 20th, extreme

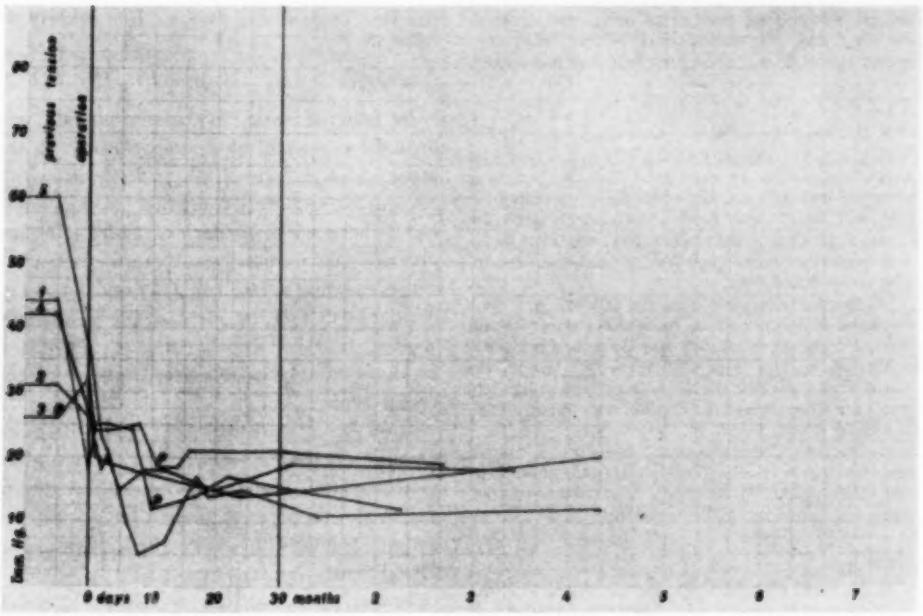


Chart 3 (Urrets-Zavalía). CHRONIC SIMPLE OR CONGESTIVE GLAUCOMA OF AN IMMEDIATE FAVORABLE PROGNOSIS. Each individual curve represents the tensional variations of one of the observations referred to in the text. (P) Tension under pilocarpine.

miosis was present and the tension was 18 mm. Hg. Vision was 20/25, corrected.

In spite of the tension being normalized by miotics, it was decided to perform diathermia retrociliaris of low intensity for the reason that the patient lived at a great distance and could not be seen frequently. This procedure was carried out on April 26th. Preoperative tension, under pilocarpine, was 18 mm. Hg; postoperative, 7.0 mm. Hg.

On the next day, the right eye presented slight conjunctival congestion and the pupil was vertically oval, dilated, displaced nasally, and almost inert. Tension was 25 mm. Hg; corrected vision, 20/50.

By May 4th, the pupil of the right eye was central, round, and mobile. Tension was 24 mm. Hg; corrected vision, 20/25. One-percent pilocarpine, three times a day, was prescribed. At examination on July 2nd, tension of the right eye was 12 mm. Hg; corrected vision, 20/20. The visual field was unchanged.

Since the hypotensive effect of the operation was insufficient, this eye has to be kept under miotics.

CASE 2

F. A., aged 65 years, was seen on December 9, 1950, with narrow-angle chronic simple glaucoma of the right eye. There was moderate cupping and pallor of the disc. The 24-hour base pressure was 31 mm. Hg (Schiøtz). Corrected vision was 20/60. The visual field was moderately constricted. One-percent pilocarpine, six times daily, was prescribed.

On April 21, 1951, tension in the right eye, under pilocarpine, was 26 mm. Hg; corrected vision was 20/30; the visual field had become even narrower.

Diathermia retrociliaris of low intensity was performed on the right eye on April 25th. The preoperative tension, under pilocarpine, was 26 mm. Hg; postoperative, 25 mm. Hg. On April 27th, conjunctival congestion and slight chemosis were present. The tension was 18 mm. Hg; corrected vision, 20/30.

By August 7th, the tension of the right eye was 18 mm. Hg; the vision corrected to 20/30; and the visual field showed no further changes. Foci of choroidal and retinal atrophy were seen at the points where the electrode had been applied.

CASE 3

G. L., aged 68 years, was seen June 30, 1950, with narrow-angle chronic congestive glaucoma of the left eye. There was slight excavation of the disc without pallor. Tension, under pilocarpine, was 26 mm. Hg (Schiøtz). Corrected vision was 20/25. The visual field was slightly constricted.

Diathermia retrociliaris of low intensity was performed on the left eye on April 25, 1951. Preoperative tension was 32 mm. Hg; postoperative, 11 mm. Hg. On the next day, the eye showed slight chemosis and a small subconjunctival hemorrhage. Tension was 23 mm. Hg.

By May 2nd, the chemosis had disappeared. Tension was 25 mm. Hg; corrected vision, 20/30. One-

percent pilocarpine, four times daily, was ordered. By July 2nd, the tension was 19 mm. Hg; corrected vision, 20/25; the visual field was unchanged.

CASE 4

R. A., aged 58 years, was seen on April 16, 1951, with glaucoma capsulare of the left eye. There was slight excavation of the disc. The 24-hour base pressure was 42 mm. Hg (Schiøtz); corrected vision was 20/20, with moderate constriction of the visual field. One-percent pilocarpine, six times daily, was prescribed. On April 18th, tension was 30 mm. Hg, under pilocarpine.

Diathermia retrociliaris of the left eye was done on April 19th, current of moderate intensity being used. Preoperative tension was 26 mm. Hg; postoperative, 22 mm. Hg. On April 20th, there was slight chemosis and the pupil was large, sluggish, and somewhat irregular. Tension was 20 mm. Hg; corrected vision, 20/30.

By August 27th, the pupil was central, round, and reacted normally. Tension was 20 mm. Hg; corrected vision, 20/20. There was an unexpected, clear improvement in the visual field.

CASE 5

S. D., aged 62 years, was seen on April 17, 1950, with narrow-angle chronic congestive glaucoma of the right eye. There was moderate cupping of the disc; tension was 60 mm. Hg (Schiøtz); uncorrected vision, 20/80. Two-percent pilocarpine, six times a day, was prescribed.

On February 22, 1951, tension in the right eye was 30 mm. Hg, under pilocarpine, and uncorrected vision was 20/25, with marked constriction of the visual field.

Particularly intense diathermia retrociliaris was performed on April 4, 1951. Preoperative tension, under pilocarpine, was 37 mm. Hg; postoperative, 26 mm. Hg. On the next day, the right eye showed chemosis and minute conjunctival hemorrhages. The anterior chamber was filled with a plasmoid, fibrinous exudate, and tension could not be measured.

By April 6th, the right eye showed chemosis, pronounced corneal hypesthesia, the anterior-chamber exudate was more fluid, and numerous floating cells and many fine posterior synechias were present. The pupil was slightly irregular and inert. Tension was 23 mm. Hg. One-percent homatropine ointment was prescribed.

The condition was unchanged on April 10th; in addition, there were fine, pigmented keratic deposits. The depth of the anterior chamber had increased remarkably. Tension was 12 mm. Hg. By May 5th, the iridociliary reaction was less noticeable but there were many posterior synechias. The anterior chamber was now of normal depth. There was, however, an inferior marginal ulcer of the cornea. Occlusion, xeroform ointment, and cortisone drops were prescribed.

When seen on August 14th, there were multiple, fine epithelial lesions of the cornea but no iridociliary reaction. Tension was 14 mm. Hg; uncorrected vision, 20/200.

COMMENT

In four of these five cases, tension was brought to normal by cyclodiathermy. In the three cases (Cases 1, 2, and 4) in which current of moderate intensity (50 ma.) was used, there were no corneal alterations and no iridociliary reactions. Treatment neither caused an immediate constriction of the visual field (in fact, in one case the visual field showed marked improvement), nor did it produce an appreciable modification of the visual acuity.

In Case 5, in which current of high intensity (80 ma., approximately) was used, a neuroparalytic type of keratitis was produced and there was a violent iridociliary reaction, followed by a marked decrease in visual acuity.

In the remaining case (Case 3) in which current of very low intensity (30 to 40 ma.) was employed, the result was insufficient, and it was necessary to prescribe pilocarpine after some days, for the tension increased and almost reached preoperative levels.

4. GLAUCOMA OF VARIOUS TYPES WITH VERY POOR PROGNOSIS (Chart 4)

CASE 1

A. M., aged 68 years, was seen on April 18, 1951, with anterior hemorrhagic glaucoma of the left eye. There was edema of the cornea and swelling of the iris. The fundus could not be seen clearly. Tension was 53 mm. Hg (Schiøtz). Vision was hand movements at 30 cm. There was very intense ocular and periocular pain.

Since the case was so grave and it was impossible to do anything else, it was decided to perform diathermia retrociliaris on the left eye on April 27th. Preoperative tension was 90 mm. Hg; postoperative, 71 mm. Hg. On April 30th, the condition of the left eye was unchanged, the tension was 60 mm. Hg, and the vision, light perception.

Because of the persistence of intense pain, a retrobulbar injection of alcohol was given on May 17th.

CASE 2

C. D., aged 26 years, was seen on June 27, 1949, with congenital glaucoma with hydrophthalmia of both eyes. The condition was much worse in the right eye, however. There was pallor and marked

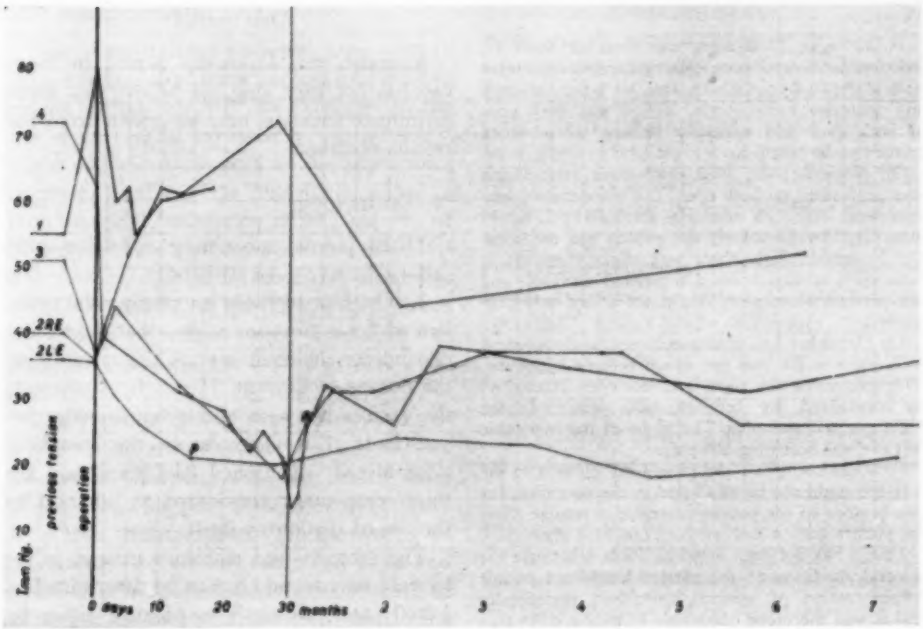


Chart 4 (Urrets-Zavalía). MISCELLANEOUS GLAUCOMAS OF VERY POOR PROGNOSIS. Each individual curve represents the tensional variations of one of the observations referred to in the text. (P) Tension under pilocarpine.

excavation of the disc in both eyes. Tension under pilocarpine was: R.E., 40 mm. Hg (Schiøtz); L.E., 36 mm. Hg. Vision was: R.E., 1/50, corrected, with wandering fixation; L.E., 20/100, corrected. The visual field of the right eye could not be plotted and that of the left eye was extremely narrow. The patient had had rather irregular miotic treatment. A progressive decrease in vision had been noticed.

Diathermia retrociliaris was performed on the right eye on October 17, 1950; and on the left eye on November 14th. There was a progressive decrease in the ocular tension in both eyes, but eventually that in the right eye increased even though pilocarpine had been resumed. On the contrary, tension in the left eye has been maintained within normal values with the aid of pilocarpine.

During the postoperative course, a pronounced chemosis and a few floating cells were observed in the anterior chambers of both eyes. By June 21, 1951, both eyes appeared the same as preoperatively. Tension, under pilocarpine, was: R.E., 26 mm. Hg; L.E., 21 mm. Hg. Vision was: R.E., 1/50, corrected; L.E., 20/70, corrected. The visual field of the left eye had become slightly narrower.

CASE 3

C. M., aged 53 years, was seen on August 28, 1947, with wide-angle, absolute glaucoma of the right eye. There was very marked cupping and discoloration of the disc. Tension was 51 mm. Hg (Schiøtz). Vision was 0/0. The left eye of this patient has already been described as Case 1 of Group 2.

On October 13, 1950, diathermia retrociliaris was performed on the right eye. Preoperative tension was 37 mm. Hg; postoperative, 37 mm. Hg. On the next day, the right eye showed slight conjunctival injection, but no iridociliary reaction. Tension was 40 mm. Hg. On November 2nd, tension of the right eye was 28 mm. Hg; on August 20, 1951, 25 mm. Hg.

CASE 4

G. L., aged 57 years, was seen on October 21, 1950, with an extremely painful absolute glaucoma of the right eye. There was corneal edema, ciliary injection, a very shallow anterior chamber. Tension was 72 mm. Hg; vision, 0/0.

Diathermia retrociliaris was performed on the right eye on October 25th, immediately preceded by a retrobulbar injection of 25-percent alcohol in two-percent novocain-adrenalin. Preoperative tension was 62 mm. Hg; postoperative, 62 mm. Hg. On the next day there was marked chemosis, the corneal edema was less, and tension was 40 mm. Hg.

By May 2, 1951, there was no pain, congestion, or corneal edema. Tension was 52 mm. Hg.

CASE 5

P. M., aged 19 months, was seen on April 13, 1949, with bilateral congenital glaucoma associated with slight hydrophthalmia, anterior polar cataract, and posterior meningocele. Vision was very poor in both eyes and naturally became worse when miotics were instilled.

On November 22, 1950, diathermia retrociliaris was performed on both eyes. The preoperative tension was: R.E., 26 mm. Hg (Schiøtz); L.E., 44 mm. Hg. Postoperatively the tension was the same. On November 25th, there was slight chemosis in both eyes, tension seemed somewhat elevated, and one-percent pilocarpine, five times a day, was prescribed.

On December 1st, spontaneous retinal detachment took place in the left eye which became hypotonic. The tension of the right eye, however, seemed to be normalized. By July 24, 1951, tension in the right eye was two-plus. The globe of the hypotonic left eye was becoming atrophic.

It would seem that the retinal detachment in the left eye could not be attributed to the operation for one brother of the patient presented a similar clinical picture and he had not been operated upon.

Case 5 has not been listed in Table 4 because the general condition of the patient would not permit administration of general anesthesia periodically and it was therefore impossible to measure the tension accurately.

COMMENT

In one patient in Group 4 (Case 1) diathermia retrociliaris did not reduce the tension nor alleviate the pain. In one of the two cases of absolute glaucoma (Case 4) tension showed only slight decrease; in the other (Case 3) it showed marked and sustained decrease, although it always remained a little above normal.

In one case of congenital glaucoma with great visual deterioration (Case 2) tension was reduced in both eyes, with the aid of pilocarpine, although in only one of them, the better, to entirely normal values. The vision in this better eye suffered no further decrease but the visual field became more restricted.

Operation in the other case of congenital glaucoma (Case 5) was a complete failure for the tension could not be modified.

In only one case (Case 2) were cells observed floating in the anterior chamber. In three cases (Cases 2, 4, and 5) chemosis of varying degrees was observed.

DISCUSSION

Although the follow-up period in these cases is not very long, we believe that some definite conclusions may be drawn from the results obtained.

A. TECHNIQUE

Three points concerning operative procedure deserve to be stressed:

1. The first of these is—putting the problem of the indications aside—that the results depend for the most part on the accuracy of the dosage of current. If this is insufficient, the results are also and proportionally insufficient; if it is excessive, the complications already mentioned take place, and are then erroneously considered as inherent to the use of diathermy itself.

The security and efficiency margin is relatively narrow and has to be determined as exactly and cautiously as possible either by means of personal experience or by the use of a thermometric electrode, which does not, however, exclude experience.

It is wise in every case to employ currents of a low intensity at the beginning, rather risking the possibility of having to repeat the operation, if it is ineffective, than to produce a harmful cauterization.

2. The second important factor is the use of a blunt electrode. This makes it impossible to say, when speaking of diathermia retrociliaris, what Benner[5] recently wisely affirmed about Vogt's operation—that its risks are still serious, and that it continues to be an intervention threatened by numerous complications.

3. The third fundamental point is that the applications be performed at not less than seven mm. from the limbus, a condition on which, in a great measure, its almost absolute harmlessness depends.

We consider chemosis of varying degrees, as well as slight and transient mydriasis accompanied by a certain degree of pupillary inertia, as phenomena of normal occurrence in the postoperative course.

B. Complications

The complications observed have already been mentioned by other authors and are due almost exclusively—in our opinion—to the use of current of excessively high intensity. Some complications, such as the appearance of floating cells in the anterior chamber, a thin marginal infiltration of the cornea, or fine ulcerations of the cornea, are not serious. Others, such as an extensive ulceration with hypesthesia of the cornea or serious iridociliary reactions with formation of pupillary synechias, are indeed of prime importance.

A decrease of vision, nearly always transitory, was observed in several cases, but not an abrupt constriction of the visual field, as sometimes happens after other operations. It is true that ultimately the narrowing of the central isopters has continued in several cases. We feel, however, that it cannot be attributed to the operation itself, but to the vascular lesions which constitute the cause of glaucoma. Burke[6] points out that, after diverse antiglaucomatous operations performed with favorable results, a progressive and inexorable restriction of the visual field takes place in 50 percent of the cases.

On the other hand, we have never encountered hyphema or hemorrhages in the vitreous as Reiser[25] did, nor even those other more serious complications already mentioned when reference was made to original cyclodiathermy.

C. Indications

Our results are broadly comparable to those obtained by Weekers,[52, 53] Verrey,[34, 35] and Reiser[25] with Weekers' procedure, and to those recently attained—though with a greater risk—by Benner[5] and Sautter[27] with Vogt's technique.

Our own experience—limited, it is true—and that of the aforementioned authors convinces us that diathermia retrociliaris is the operation of choice in nearly every form of secondary glaucoma; in many cases it is an irreplaceable surgical procedure. If it proves insufficient, it can be repeated without difficulty one or several times, or followed by another procedure. Verrey[35] points out the fact that in glaucoma secondary to keratoplasty the results are particularly good; we have not yet had an opportunity of applying the procedure in such a case.

The same may be said of those cases of genuine chronic glaucoma and of infantile or juvenile glaucoma with severe damage to visual function. In those of our patients operated upon with adequate current, further restrictions of the visual field which could be ascribed directly to the operation have not been observed.

In incipient cases of favorable prognosis, we believe with Sautter[27] that preference must be given to the classical operations—Lagrange's or Elliot's iridosclerectomy, or iridencleisis for chronic glaucoma, goniotomy or iridencleisis for congenital glaucoma. These procedures reduce more definitely the abnormally elevated tension. Diathermia retrociliaris does not expose the patient to more complications than the other operations (Benner[5]). Verrey,[35] as well as Sautter,[27] has stressed the fact—and it is confirmed by our experience—that the tension may undergo a new increase some months after cyclodiathermy has been performed.

To our knowledge, the same criterion may be applied to cases of acute glaucoma, although we have not had the opportunity of using this operation for acute glaucoma. Verrey[34] and Desvignes and Naudin[9] have employed diathermia retrociliaris with success as a first operation in some such cases, but it seems more prudent to resort in the first place to iridectomy or to iridencleisis (Vogt,[42, 43] Benner[5]).

According to the observations of Weekers[55] and Sautter,[27] we do not hesitate, however, to consider diathermia retrociliaris as the best complementary operation in almost every case in which a previous intervention has been ineffective.

The circumstance, pointed out by Reiser,[25] that, in the cases in which this operation was

performed, the provocative tests did not succeed in determining any significant tension increase, is particularly remarkable.

As to the cases of absolute or hemorrhagic glaucoma, we venture to say that, though diathermia retrociliaris often proves useless, it is advisable to perform it, after a retrobulbar injection of alcohol is given, since it is the only operation which has some chance of being successful.

Conclusions

Indications for the use of diathermia retrociliaris are more or less the same as those for original cyclodiathermy. Its dangers are, however, much smaller.

Between these two operations there are, however, no essential differences. They are —as we shall point out in a future publication—only quantitatively different. The results attained, as well as the studies performed in enucleated eyes, show that their mode of action is basically the same.

Weekers' technique is more simple, more effective, and more secure because only blunt electrodes are used, because from the very first the whole ciliary body is affected and, above all, because the applications are located behind the ciliary body, so as to damage the fibers by which it is supplied, without inflicting on it a direct injury.

27 de Abril 255.

References

1. Albaugh, C. H., and Dunphy, E. B.: Arch. Ophth., **27**:543, 1942.
2. Amsler, M.: Arch. d'opht., **1**:856, 1937.
3. Arato, I.: Arch. Soc. oftal. hispano-am., **9**:746, 1949.
4. Arruga, H.: Arch. Soc. oftal. hispano-am., **10**:224, 1950.
5. Benner, R.: Ann. d'ocul., **180**:89, 1947.
6. Burke, J. W.: Am. J. Ophth., **23**:657, 1940.
7. Coppez, L.: Ophthalmologica, **109**:80, 1945.
8. Curran, E. J.: Arch. Ophth., **54**:321, 1925.
9. Desvignes, P., and Naudin: Arch. d'opht., **8**:589, 1948.
10. Díaz-Domínguez, D.: Arch. Soc. oftal. hispano-am., **8**:117, 1948.
11. Duke-Elder, W. S.: Am. J. Ophth., **33**:11, 1950.
12. Fiore, T.: Zentralbl. f. d. ges. Ophth., **22**:780, 1930.
13. Franceschetti, A.: Arch. d'opht., **1**:857, 1937.
14. François, J.: Ann. d'Ocul., **182**:169, 1949.
15. ———: Ophthalmologica, **121**:1, 1951.
16. Gasteiger, H.: Klin. Monatsbl. f. Augenh., **107**:52, 1941.
17. Guerry, D.: Am. J. Ophth., **27**:1376, 1944.
18. Kettesy, A.: Ophthalmologica, **120**:334, 1950.
19. Marr, W. G.: Am. J. Ophth., **32**:241, 1949.
20. Meesmann, A.: Klin. Monatsbl. f. Augenh., **109**:721, 1943.
21. Meyer, S. J.: Arch. Ophth., **41**:417, 1949.
22. Moreu, A.: Arch. Soc. oftal. hispano-am., **6**:468, 1946.
23. Palomar, A. P.: Arch. Soc. oftalm. hispano-am., **4**:608, 1944.
24. Redslob, E.: Ann. di ottal. e clin. ocul., **72**:449, 1946.
25. Reiser, K. A.: Klin. Monatsbl. f. Augenh., **115**:491, 1949.
26. Richner, H.: Schweiz. med. Wchnschr., **70**:269, 1940.
27. Sautter, H.: Klin. Monatsbl. f. Augenh., **115**:481, 1949.
28. Stocker, F. W.: Arch. Ophth., **34**:181, 1945.
29. Stratton, D.: Am. J. Ophth., **31**:239, 1948.
30. Streiff, E. B.: Klin. Monatsbl. f. Augenh., **101**:910, 1938.
31. Thiel, R.: Klin. Monatsbl. f. Augenh., **109**:744, 1943.
32. Urrets-Zavalía Jr., A.: Ophth. ibero am., **10**:1, 1948.
33. ———:Ophthalmologica, to be published.
34. Verrey, F.: Ophthalmologica, **117**:281, 1949.
35. ———: Ophthalmologica, **121**:101, 1951.
36. Villaseca, A.: Arch. Chilenos oftal., **4**:425, 1947.
37. Vogt, A.: Schweiz. med. Wchnschr., **66**:593, 1936.
38. ———: Klin. Monatsbl. f. Augenh., **97**:672, 1936.
39. ———: Klin. Monatsbl. f. Augenh., **99**:9, 1937.
40. ———: Schweiz. med. Wchnschr., **67**:367, 1937.

41. ———: Klin. Monatsbl. f. Augenh., **103**:591, 1939.
42. ———: Arch. d'opht., **3**:1071, 1940.
43. ———: Klin. Monatsbl. f. Augenh., **106**:632, 1941.
44. Wagner, H.: Klin. Monatsbl. f. Augenh., **101**:909, 1938.
45. ———: Klin. Monatsbl. f. Augenh., **104**:344, 1940.
46. ———: Klin. Monatsbl. f. Augenh., **106**:231, 1941.
47. ———: Klin. Monatsbl. f. Augenh., **108**:509, 1942.
48. Wagner, H., Karbacher, P., Meyer, C., Wolf, H., and Weber, E.: Klin. Monatsbl. f. Augenh., **107**:457, 1941.
49. Wagner, H., and Richner, H.: Schweiz. med. Wchnschr., **69**:1048, 1939.
50. Weekers, L.: Ophthalmologica, **118**:564, 1949.
51. Weekers, L., and Weekers, R.: Ophthalmologica, **104**:1, 1942.
52. ———: Ophthalmologica, **109**:212, 1945.
53. ———: Acta Ophth., **24**:1, 1946.
54. ———: Ann. d'ocul., **180**:76, 1947.
55. ———: Ann. d'ocul., **182**:188, 1949.
56. Weekers, L., Weekers, R., and Roussel, F.: Ophthalmologica, **117**:65, 1949.
57. Weve, H. J. M.: Nederl. tijdschr. v. geneesk., **76**:5335, 1932.
58. ———: Nederl. tijdschr. v. geneesk., **77**:1947, 1933.

SYMPATHETIC OPHTHALMIA FOLLOWING CYCLODIATHERMY*

MARTIN BODIAN, M.D.
Brooklyn, New York

Sympathetic ophthalmia rarely follows cyclodiathermy. Since the operation was first described in 1936 by Vogt,[1] it has been repeated with modifications many hundreds of times. To the present day, four cases of sympathetic ophthalmia following cyclodiathermy have been mentioned in the literature.

One of these eyes had had a perforating injury with iridocyclitis prior to surgery. Cyclodiathermy was done to relieve secondary glaucoma. Alan Woods, who communicated the report of this case to Berens,[2] is of the opinion that the original injury was the more likely cause of the sympathetic ophthalmia.

The three remaining cases to follow cyclodiathermy are perfunctorily presented in the literature.

In 1938, in a discussion of Wagner's[3] paper on cyclodiathermy, Streiff[4] tersely noted that, following Vogt's operation, an eye was found to have the pathologic anatomy of sympathetic ophthalmia. Sautter[5] strongly doubts that cyclodiathermy caused sympathetic ophthalmia in Streiff's case for the following reasons: (1) The uveitis was not binocular and, therefore, was probably not a sympathetic inflammation; (2) uveitis was present in this eye prior to the application of cyclodiathermy.

In France, Thomas and others[6] mentioned one instance of sympathetic ophthalmia which followed 100 perforating cyclodiathermy operations. No details of this case are present in their article.

The last plausible case of sympathetic ophthalmia following cyclodiathermy to appear in print is quoted by Berens.[2] He reports a personal communication from Runge on the development of sympathetic ophthalmia six weeks after nonperforating diathermy for absolute glaucoma. As in the previous case, no mention is made of the preceding history which led to the glaucoma. Unlike the previous cases, this is the first instance to follow a nonperforating procedure.

One is impressed by the large number of cyclodiathermy operations which were not followed by sympathetic ophthalmia.

---

* From the Brooklyn Eye and Ear Hospital and the Long Island College Hospital in Brooklyn.

Almost every author on the subject has been cognizant of the fact that trauma of the ciliary body is an important forerunner of sympathetic ophthalmia. In view of this fact, it is surprising that so few cases of this complication have followed diathermy to the ciliary body.

DeWecker is quoted by Vogt[7] to indicate that after hundreds of diathermy-puncture operations to the sclera for retinal detachments not a single case of sympathetic ophthalmia had arisen. As is well known, uveal tissue of the choroid receives massive injuries in these operations.

Vogt[8] further reported in his earliest summary of cases that sympathetic ophthalmia was conspicuously absent among the complications of the operation. He was further encouraged by the benign effects of this operation when he applied it to a case of secondary glaucoma which had had an antecedent uveitis of possible sympathetic origin. In this case, not only did the glaucoma abate but the uveitis cleared up as well. By 1941, he was able to report on over 100 cases of cyclodiathermy. Not one was complicated by sympathetic ophthalmia.[7]

Wagner[9] published the results of 200 cyclodiathermy operations performed at the University of Zurich in 1941. After recommending the operation, he notes that sympathetic ophthalmia was not observed in any of their cases.

In America, Albaugh and Dunphy[10] described their technique of surface diathermy for the first time in 1942. Pathologic study of two eyes that were enucleated following cyclodiathermy showed widespread destruction to the ciliary body. In spite of the trauma inflicted, sympathetic ophthalmia did not occur in any of the 32 cases which they reported.

On the basis of a questionnaire sent to several German eye clinics, Thiel[11] collected data on 336 cases that had had cyclodiathermy operations. No case of sympathetic ophthalmia could be found among these.

By 1949 Meyer[12] was able to tabulate the findings on 126 cyclodiathermy operations. No case of sympathetic ophthalmia was present among these.

By far the most impressive statistics on follow-up after cyclodiathermy operations were compiled by Berens.[2] In discussing a paper by Bietti, he presented the results of a questionnaire sent to members of the American Ophthalmological Society. Eighty eye surgeons responded. There were 201 nonperforating and 363 perforating operations, a total of 564. To this number he was able to add 193 results of cyclodiathermy operations performed at the New York Eye and Ear Infirmary. His grand total was 757 studies.

Among these was the one case of sympathetic ophthalmia presented by Runge already referred to. In another case of sympathetic ophthalmia submitted in this group, the patient had suffered severe trauma previous to the operation. Because of this, the sympathetic ophthalmia must be excluded as having been produced by cyclodiathermy. An interesting sidelight on Berens' findings was that more complications, such as iritis and phthisis bulbi, were to be found following the nonperforating than the perforating cyclodiathermy.

From the foregoing, it can be seen that only three cases of sympathetic ophthalmia, not all of which have been verified, have followed well over a thousand cyclodiathermy operations. With a view toward determining whether any cases had been omitted because of oversight, the literature on sympathetic ophthalmia was reviewed as far back as 1936 when the first reports on cyclodiathermy were issued.

In no instance was I able to find cyclodiathermy listed as a cause of a sympathetic ophthalmia in any cases except those already mentioned. It must be added, however, that almost every author writing on the subject of cyclodiathermy has been aware that this operation might produce sympathetic ophthalmia.

Joy,[13] in 1949, published an analysis of

436 proved cases of sympathetic ophthalmia. After checking through the various etiologic agents, he was unable to find cyclodiathermy among them. He comments by way of explaining this absence of cases that the reaction to diathermy is different from that to other operations. Diathermy causes the openings to close almost immediately and the tissues tend to shrink away from the wound because of their elasticity. From his results and this reasoning he concludes that cyclodiathermy is an extremely safe operation so far as sympathetic ophthalmia is concerned.

The same year, McPherson[14] reviewed all the cases of sympathetic ophthalmia that had occurred at the Wilmer Institute in Baltimore between 1927 and 1948. He was able to cull 61 undoubted instances of sympathetic ophthalmia. Cyclodiathermy was not mentioned as a possible cause in any of these cases. His compilation further showed that 59 percent of the sympathetic ophthalmias in his series were due to penetrating wounds of the anterior uveal tract.

Case presentation

Y. R., a 63-year-old white housewife, was first seen on August 23, 1949, complaining of loss of vision in her left eye for one week.

Past history. The patient had been obese for many years. No attempts at weight control had been made. Her blood pressure had been high but variable, with frequent rises to 180 mm. Hg, systolic. Aside from this, she had had no serious illnesses.

Family history. One sister is being treated for glaucoma simplex. The tension in both eyes has remained at the upper level of normal. Other family history was noncontributory.

Present illness. One week before her first visit, the patient woke up and noted that the vision in the left eye was poor. She had a feeling that objects were double when seen with this eye. There was no attendant pain or other subjective symptoms. No halos were noted around lights. The condition had not changed for better or worse since its onset. No form of treatment had been used.

Physical examination. The patient was an obese white woman in no apparent distress. Temperature was 98.6°F., pulse 72, respiration 20, blood pressure 184/96 mm. Hg. The skin showed no obvious disease. Her head was normal in size and shape. Ears, nose, and throat were free of pathologic findings. The heart was slightly enlarged to the left, regular sinus rhythm, with a forceful apex beat; no thrills or murmurs. The lungs were clear to auscultation and percussion. The abdomen was obese but there was no tenderness or visceral enlargements. The extremities presented no swellings, tenderness, or edema.

Eye examination. Right eye. Vision was 20/30−2. The lids, conjunctiva, cornea, anterior chamber, pupils, iris, and lens showed no pathologic condition. The fundus showed moderately advanced copper wiring of the arteries and arteriovenous compression. No hemorrhages or exudates were visible. The disc was of normal color, sharply outlined and had a physiologic cup. Tension equaled 28 mm. Hg (McLean).

Left eye. Vision, counts fingers at three feet. The lids, conjunctiva, cornea, pupils, iris, lens show no pathologic condition. The fundus showed the typical picture of central retinal vein occlusion. The branch veins were enormously distended and tortuous. Hemorrhages of all sizes and shapes were spread over the entire fundus. The arteries were somewhat attenuated. The disc was completely blurred with hemorrhage and edema, so that no details could be seen. Tension equaled 35 mm. Hg (McLean).

Eye muscles. Extraocular motility was normal, convergence-power fair, exophoria for near equaled nine prism diopters with orthophoria for distance.

Course. The patient was given pilocarpine (1.0 percent) eyedrops for the left eye three times a day as a prophylaxis against glaucoma. Five drops of saturated solution

of potassium iodide, in one-half glass of water, were prescribed three times a day, after meals. The patient noted no change in her condition until September 19, 1949, just five weeks after the onset of her symptoms.

At this time she had a sudden onset of pain in the left eye which radiated to the left side of her face and head. Vision was reduced to light perception. This was accompanied by redness of the eye and much lacrimation. In spite of these symptoms, she did not come to my office until one week later.

At this time she was noted to have all the signs and symptoms of an acute congestive glaucoma in the left eye. This included redness and swelling of the lids, congestion and chemosis of the conjunctiva, edema of the cornea, and a dilated pupil. The fundus reflex was lacking and no structures of the retina could be made out. The tension in the left eye was 110 mm. Hg (McLean).

The right eye was as seen on the original physical examination, except for the tension. This had risen to 55 mm. Hg (McLean).

DFP (Floropryl) was instilled into both eyes. Pilocarpine (2.0 percent) was ordered to be used for the right eye three times a day and for the left eye every two hours.

When she returned the next day, the left eye still presented all the features of acute glaucoma except that the pupil had diminished to 2.5 mm. The tension in this eye was 100 mm. Hg (McLean). The pupil in the right eye had become 1.5 mm. and the tension had fallen to 38 mm. Hg (McLean). The patient was instructed to use furmethide (10-percent solution) in the left eye, every two hours.

The following day the tension was still 100 mm. Hg (McLean) in the left eye, and the patient was hospitalized for cyclodiathermy and posterior sclerotomy operations. These were done on October 10, 1949, six weeks after the central vein occlusion and four days after the onset of acute congestive glaucoma.

Surgical procedure. The lower conjunctiva was incised in a curved line parallel to the limbus, eight mm. behind the cornea. A double row of surface diathermy applications was made to the sclera beginning three mm. from the limbus. Among the surface applications, about six perforations were made through the sclera with a 0.5-mm. diathermy needle. A sclerotomy was made with a two-mm. Elliot trephine, 13 mm. behind the limbus at about the 4-o'clock position.

The exposed choroid was perforated with a diathermy needle to permit the release of a small bead of vitreous. This was done to prevent the secondary rise in tension that frequently follows cyclodiathermy.

The postoperative course was quite stormy. There was much redness and swelling of the eye, as well as severe pain. At the first dressing, 24 hours after operation, a small hemorrhage was seen in the anterior chamber.

The tension remained high to finger palpation for the next six weeks. It then began to subside, with the onset of deep uveitis. By November 26, 1951, eight weeks following operation, the left eye had begun to show evidence of atrophy. The iris could be seen bound down to the lens. The anterior chamber was cloudy. The cornea showed vascularization and clouding over the lower fourth.

In the meantime, the tension in the right eye had shown poor control with pilocarpine. It varied between 34 and 75 mm. Hg (McLean). DFP (Floropryl), eserine, furmethide, and other miotics lost their effectiveness one by one. Visual fields for the right eye remained normal, in spite of these rises. The optic disc showed no glaucomatous cupping.

On January 28, 1950, almost four months after her operation, several small keratic precipitates were seen in the right eye. However, there was no aqueous flare. Vision at this time in the right eye was 20/40 and correctible with a pinhole to 20/20−3. The pupil was three mm., in spite of miotics, and fixed to light. The tension was 75 mm. Hg

(McLean). Visual fields were normal.

The left eye at that time showed advancing phthisis bulbi with no light perception. Tension was 18 mm. Hg (McLean). A low grade uveitis continued to smolder.

Because of the apparent onset of sympathetic ophthalmia in the right eye and the hopeless destruction of the left, enucleation of the latter was decided upon after consultation with Dr. Mortimer A. Lasky. This was done on February 2, 1950.

Surgical procedure. After a limbal circumcision of the conjunctiva, the extraocular muscles were isolated and severed from the globe. This was then freed from surrounding tissues. The optic nerve was engaged by a tonsillar snare and severed. A glass-ball implant was placed in the muscle cone. Tenon's capsule was closed over this. The opposing rectus muscles were sutured to each other. The conjunctiva was closed along a horizontal line with a black-silk continuous suture.

The postoperative course was smooth. The tissues healed without difficulty and it was possible to place a prosthesis in the socket 17 days after enucleation. The tension in the right eye responded to miotics and fell to 40 mm. Hg (McLean) within two weeks.

The keratic precipitates began to diminish in number and size, and within three months showed only a few dustlike, pigmented particles. The vision improved to 20/20+2 and could be further corrected to 20/15 with a plus 0.5D. sph.

Since that time tension has ranged between 38 and 45 mm. Hg (McLean) in the right eye. The pupil is kept to 1.5 mm. by the use of 1.5-percent pilocarpine solution three times a day. There has been no loss of visual field. The fundus shows only the retinal arteriosclerosis noted on the first examination.

When last seen on December 8, 1951, 22 months after the left eye was enucleated, the vision in the right eye was correctible to 20/15, the tension was 40 mm. Hg (McLean), and there was no evidence of uveitis.

Pathologic findings in enucleated eye (figs. 1, 2, 3, and 4)

Cornea

Lower half of corneal stroma is thickened to three times normal size. This area is vascularized and infiltrated with leukocytes. These cells show a marked perivascular distribution. The upper and lower limbus is heavily infiltrated with small round cells, epithelioid cells, and occasional eosinophils.

Sclera

The region adjoining the lower portion of the cornea which was treated by diathermy appears necrotic. Its width is shrunken to half its thickness.

There is complete absence of fixed cells. The remainder of the sclera is infiltrated with round cells characteristic of sympathetic ophthalmia. This infiltration has a notable perivascular distribution.

A large tangentially cut vein with a heavy cellular collar can be seen near the posterior pole of the globe.

Anterior chamber

This contains a clear albuminoid material. The filtration angle is obliterated by peripheral anterior synechias. A fibrous membrane containing leukocytes occludes the pupil and extends onto the anterior surface of the iris.

Iris

This is thickened, vascularized, and infiltrated with polymorphonuclear as well as round cells. Its architecture is in the process of disintegration. Peripheral anterior adhesion to the cornea and posterior total synechia to the lens give evidence of previous glaucoma.

Lens

This is surrounded by inflammatory and granulation tissues on all sides. Below, it is subluxated where it firmly adheres to the disorganized ciliary body. An anterior cap-

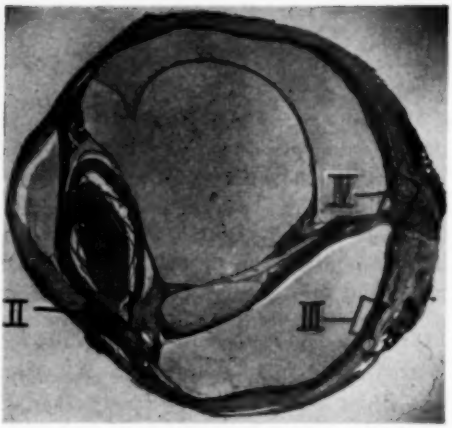


Fig. 1 (Bodian). Magnification (×7.5) of enucleated eye in presented case of sympathetic ophthalmia. The "tumified" choroid is prominent evidence of the sympathetic ophthalmia. Destruction of the sclera and ciliary body due to diathermy is present in Area II. (The Roman numerals refer to sections more highly magnified in Figures 2, 3, and 4.)

sular cataract is present in the pupillary area. The cortex shows advanced degeneration.

Ciliary body

The lower half of the ciliary body is drastically altered as a result of the diathermy procedure. The entire structure is destroyed. Muscle tissue, epithelium, and interstice are a shrunken mass, to which adheres the lens.

The pigment is accumulated in spherical masses of varying size. Some of these have migrated into the posterior chamber. Granulation tissue pervades the entire area.

The upper half of the ciliary body, which received no diathermy, contains a marked cellular infiltration, vascularization, and edema. The muscle bundles are separated by these processes.

Vitreous body

Anteriorly, a cyclitic membrane reaches across the entire retrolental space. A com-

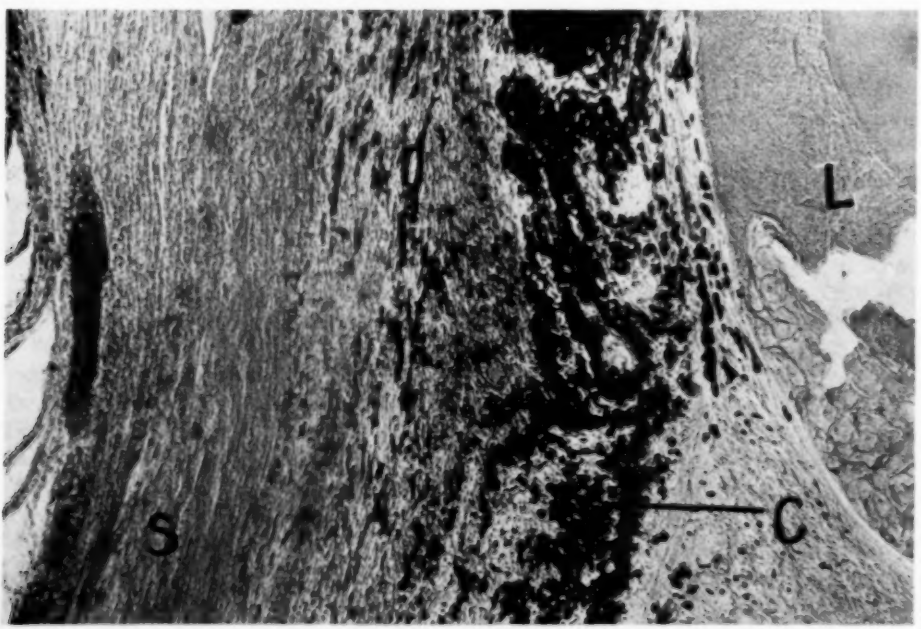


Fig. 2 (Bodian). See Figure 1, Area II. (S) Necrotic sclera, (C) disorganized ciliary body, and (L) degenerating lens in area of cyclodiathermy (×150).

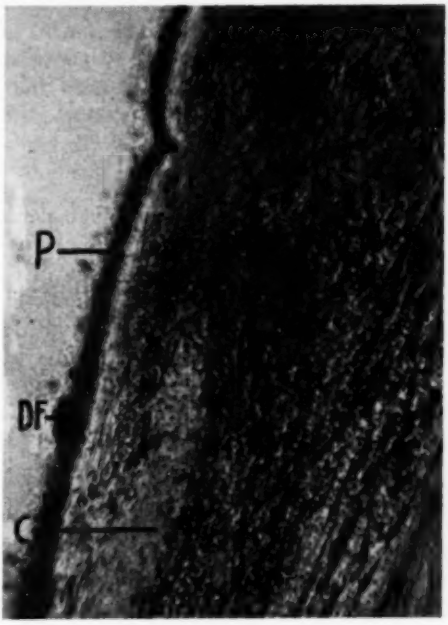


Fig. 3 (Bodian). See Figure 1, Area III. (C) Choroid is intensely infiltrated with round and epithelioid cells. (P) The pigment epithelium of the retina contains a (DF) Dalen-Fuchs nodule which is considered by many as pathognomonic of sympathetic ophthalmia (×150).

pletely detached retina extends forward from its mooring to the optic nerve. Pigment granules and leukocytes can be occasionally noted in the remaining vitreous matrix.

Choroid

This shows the typical pathologic findings of sympathetic ophthalmia. There is a tumorlike, generalized infiltration with lymphocytes, plasma, epithelioid, and giant cells. This is more marked in the posterior portion where the choroid is several times its normal thickness.

Perivascular collars of lymphocytes are numerous. Epithelioid cells are grouped in pigmented nests. These contain giant cells formed by the fusion of epithelioid elements.

Retina

This is completely detached. Degenerative changes are especially noticed in the nerve-fiber and ganglion layers. Round-cell infiltration is widespread but most marked in the perivascular areas.

The pigment epithelium presents several "Dalen-Fuchs nodules." These consist of masses of proliferated pigment cells that are undergoing autolysis and invasion by phagocytes.

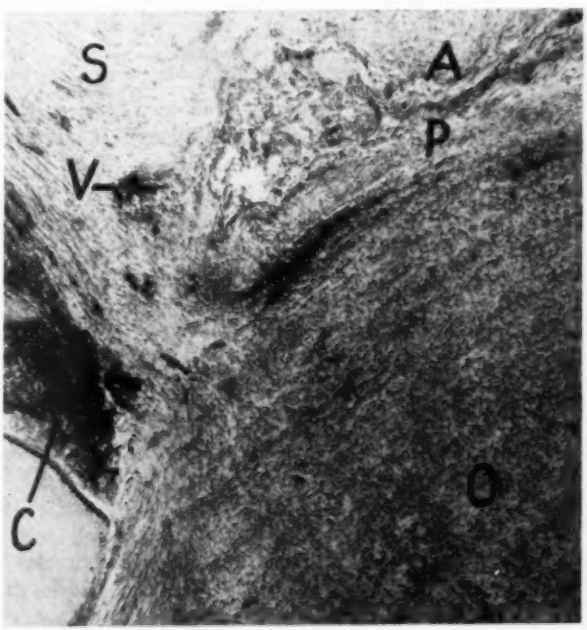


Fig. 4 (Bodian). See Figure 1, Area IV. (O) Optic nerve, (P) pia, and (A) arachnoid are diffusely infiltrated with round cells. Epithelioid and round cells are massed in the (C) choroid. Perivascular infiltration (V) typical of sympathetic ophthalmia may be seen in the (S) sclera (×150).

Optic nerve

This is heavily seeded with round cells. The pia and arachnoid show the same infiltration. Mantles of cells surround the vessels.

Discussion

The four cases of sympathetic ophthalmia following cyclodiathermy cited in the literature have already been mentioned. One case[2] was discounted by the original observer (Woods), since he felt that the sympathetic ophthalmia was due to previous trauma. A second case (Streiff) of sympathetic ophthalmia which supposedly followed cyclodiathermy[4] has been seriously challenged by Sautter[5] since there was a preëxisting uveitis.

The remaining two instances of sympathetic ophthalmia to follow cyclodiathermy are those mentioned by Berens[2] and Thomas and others.[6] These reports contain no details. Thus, not one confirmed, detailed report is published of sympathetic ophthalmia following cyclodiathermy.

Joy[13] is also impressed by the rarity of this complication. In his paper on sympathetic ophthalmia in 1949, he fails to mention a single case following cyclodiathermy. His explanation for this absence follows: "Cyclodiathermy and operations for retinal detachment do not favor sympathetic ophthalmia, for the uvea does not prolapse. The reaction causes the openings to close almost immediately and the tissues tend to shrink away from the wound because of their elasticity." In view of this statement my case becomes most unusual.

Because of the apparent healing of the uveitis in the remaining eye, some may challenge the diagnosis of sympathetic ophthalmia in the presented case. However, there are many instances reported in which cures have been effected with restoration of normal vision. S. R. Gifford[18] treated a group of 27 cases of sympathetic ophthalmia from various causes. Eight patients regained 20/20 vision, eight 20/30, two 20/50, three could only count fingers, and six could perceive hand movements or less.

As far back as 1932, Wurdemann[16] was able to gather an impressive number of cures of sympathetic ophthalmia from the literature (Thompson, Snell, Woods, Green, Burch, Verhoeff, and others). Normal vision was restored frequently among these cases.

Considering the great variety of therapeutic agents used by these authors, one is prompted to believe that nature had a hand in producing some of the results. I feel that the clearing-up of the uveitis in my patient's right eye represents a cure such as those reported.

There are further factors to substantiate the diagnosis of sympathetic ophthalmia in my patient. The time-interval between surgery and the onset of uveitis in the opposite eye was about three and one-half months. This is not an unusual incubation period for sympathetic ophthalmia. The preceding, smoldering inflammation of the exciting eye is also highly suggestive of sympathetic ophthalmia.

The most positive proof that the diagnosis of sympathetic ophthalmia is correct comes from a study of the enucleated eye. This showed the major criteria for diagnosis as stated by Duke-Elder.[17] Briefly these are: (1) Uniform infiltration of the uvea, especially the choroid; (2) perivascular round-cell infiltration; (3) presence of Dalen-Fuchs nodules; (4) absence of caseation or necrosis.

Because of the clinical as well as pathologic features of the presented case, sympathetic ophthalmia is the only diagnosis permissible.

Could the posterior sclerotomy have produced sympathetic ophthalmia? This is extremely unlikely.

In the first place, the scleral opening was made at the equator so that choroid was the only uvea involved. It is well known that choroidal trauma is not usually considered a cause of sympathetic inflammation.

This is borne out by the many thousands

of operations for detached retina which are never followed by this complication. Furthermore, many prominent authors,[13-15] writing about sympathetic ophthalmia, fail to list posterior sclerotomy as a precursor to that disease.

One further thought arises on considering this case. Is the chronic noncongestive glaucoma in the remaining eye primary or secondary? I feel that it is primary for several reasons: (1) All evidence of active iritis has subsided for over a year; (2) the tension responds to miotics; and (3) the patient has a sister with chronic simple glaucoma of almost identical proportions and response to miotics.

Summary and Conclusions

A review of the literature indicates the extreme rarity of sympathetic ophthalmia following cyclodiathermy. In only two cases is the causal relationship unchallenged. Neither of these latter reports contains the least detail as to previous eye condition, type of diathermy used, time relationships, or other pertinent information.

A case is presented which developed sympathetic ophthalmia three and one-half months following penetrating and surface cyclodiathermy. This operation had been done for hemorrhagic glaucoma which followed closure of the central retinal vein.

The clinical course, as well as the pathologic findings, are presented. These are characteristic of sympathetic inflammation. Reasons have been presented to indicate that this inflammation was caused by the preceding cyclodiathermy.

Because of its rarity, fear of sympathetic ophthalmia should not deter one from performing an indicated cyclodiathermy. On the other hand, I feel that this complication must always be kept in mind when evaluating this popular operation.

349 Eastern Parkway (16).

References

1. Vogt, A.: Essai d'abaisement de la tension intraoculaire par cauterisation diathermique du corps ciliare. Abstracted from Soc. Suisse Ophtal. June, 1936. Arch. d'ophtal., **1**:856, 1937.
2. Berens, C.: In discussion on Bietti, G.: Surgical interventions on the ciliary body. J.A.M.A., **142**:889-897 (Mar.) 1950.
3. Wagner, H.: Bisherige Ehrfahringen mit die Diathermie-stichelung des corpus ciliare bei Glaukom. Klin. Monatsbl. f. Augenh., **101**:909, 1938.
4. Streiff, E. B.: In discussion on Wagner, H.: Ibid.
5. Sautter, H.: Unsere Erfahrungen mit der Zyklodiathermiepunktur, Klin. Monatsbl. f. Augenh., **115**:487, 1949.
6. Thomas, C., Cordier, Algan, and Poirot: Resultats de cent cas de cyclodiathermie perforante de Vogt. Bull. soc. d'ophtal., Paris, April, 1948, p. 205.
7. Vogt, A.: Zyklodiathermiestichelung als Ultima Ratio. Klin. Monatsbl. f. Augenh., **106**:232, 1941.
8. ———: Ergebnisse der Diathermiestichelung des corpus ciliare (Zyklodiathermiestichelung) gegen Glaukom. Klin. Monatsbl. f. Augenh., **99**:9, 1937.
9. Wagner, H.: Zyclodiathermiestichelung bei einigen besonderen Fallen von Glaukom. Klin. Monatsbl. f. Augenh., **106**:231, 1941.
10. Albaugh, C. H., and Dunphy, E. B.: Cyclodiathermy. An operation for the treatment of glaucoma. Arch. Ophth., **27**:543, 1942.
11. Theil, R.: Vorzuge und Nachteile der Zyklodiathermiepunktur nach A. Vogt. Klin. Monatsbl. f. Augenh., **109**:744, 1943.
12. Meyer, S. J.: Diathermy cauterization of ciliary body for glaucoma. Arch. Ophth., **41**:417, 1949.
13. Joy, H. H.: Clinical aspects of sympathetic ophthalmia. Am. J. Ophth., **32**:1383 (Oct.) 1949.
14. McPherson, S. D., Jr, Sympathetic ophthalmia: A review of 61 cases. Southern M. J., **42**:120 (Feb.) 1949.
15. Gifford, S. R.: In Berens, C.: The Eye and Its Diseases. Philadelphia, Saunders, 1936, p. 744.
16. Wurdemann, H. V.: Injuries of the Eye. St. Louis, Mosby, 1932, pp. 144-146.
17. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1941, pp. 2348-2349.
18. Spaeth, E. B.: Principles and Practice of Ophthalmic Surgery. Philadelphia, Lea, 1944, pp. 731 and ff.

DI-ISOPROPYL FLUOROPHOSPHATE (DFP) (0.01 PERCENT) IN CHRONIC WIDE-ANGLE GLAUCOMA*

IRVING H. LEOPOLD, M.D., AND ALBERT F. CLEVELAND, M.D.
Philadelphia, Pennsylvania

Since the initial investigations of di-isopropyl fluorophosphate (DFP) in glaucoma (Leopold and Comroe), innumerable studies have been made with this agent in this condition (McDonald, Lebensohn, Leopold and McDonald, Dunphy, Marr, Raiford, Stone, Ehlers, Gallino and Bottini, Goedbloed, Wheeler, Weekers, and Roussel).

From a review of the reports to date, it is apparent that di-isopropyl fluorophosphate is a valuable adjunct in the medical treatment of glaucoma. In a recent review of glaucoma, Scheie has concluded that DFP is the strongest miotic agent of its type, that it is the most effective one available in counteracting homatropine and atropine, and that it is of particular value in cases in which surgical measures are to be avoided at any cost, and in cases of secondary glaucoma following cataract extraction.

Studies with this drug have been made in acute congestive glaucoma (acute narrow-angle glaucoma), in chronic simple glaucoma (chronic wide-angle glaucoma), and in chronic congestive glaucoma (chronic narrow-angle glaucoma).

It has been tried usually in concentrations ranging from 0.05 to 0.2 percent. Practically all of the studies have dealt with these concentrations. However, Raiford reported using concentrations as low as 0.005 percent successfully in a few glaucomatous eyes. It is the purpose of this paper to report the results of low concentrations of this drug in wide-angle glaucoma.

The drug has definite disadvantages, as mentioned in the original study. In the higher concentrations, 0.05 percent and upward, it is known to produce marked ciliary spasm, causing false myopia, rapidly altering accommodation, considerable browache, and headache. Occasional eyes have shown a rise in pressure, and a few instances of retinal detachment following the use of this drug have been reported. A few eyes have developed sensitivity to di-isopropyl fluorophosphate.

There is no doubt that DFP is an effective miotic. There is also no doubt that in the concentrations usually employed it produces considerable ciliary spasm. This ciliary spasm is much more marked than that found with other miotics, particularly pilocarpine. Thus its usefulness in early glaucoma would seem to be limited, particularly since these patients must use their miotic faithfully in order to control the disease process.

An agent which produces undesirable symptoms would tend to discourage an individual from using this drug regularly and faithfully.

Because of these undesirable effects, most ophthalmologists have reserved the use of DFP for aphakic glaucoma and those cases of chronic glaucoma which have failed to respond to other therapy and in which operation must be delayed.

This present study was undertaken to determine whether DFP could be used in very dilute concentration and thus produce fewer undesirable effects, particularly on the ciliary muscle, and perhaps in this lower concentration allow it to be used more widely in glaucoma. Raiford's studies along this line, in which he employed 0.005-percent and 0.01-percent DFP, suggested that in the more dilute concentrations this agent had few undesirable symptoms.

CASE MATERIAL

Seventy eyes with chronic simple glaucoma, all showing wide angles by gonios-

* From the Research and Clinical Departments of Wills Eye Hospital. Presented at the fourth annual Wills Hospital Conference, March 22, 1952.

copy, all controlled by 0.5- to 4.0-percent pilocarpine nitrate, instillations being made three to five times daily, were used in this study.

Forty of these cases were mild and showed no visual field changes at the time of institution of DFP therapy.

The remaining 30 eyes showed moderate to fairly advanced glaucoma. The visual field changes in these 30 eyes ranged from enlargement of blindspot to Bjerrum scotomas with nasal field contractions and, in the most severe and advanced cases, only a few degrees of field fixation.

In every case, diurnal fluctuations were studied, either by measuring the intraocular pressure at different times of the day on different clinic day visits, or by hospitalization of the patient and measuring the intraocular pressure every four hours throughout a 48-hour period.

All cases were tried on 0.01-percent DFP. Frequency of instillation was determined by checking the intraocular pressure at various times throughout a 24-hour period.

One other group of eight patients was treated. These consisted of patients who had failed to respond to other miotics, had chronic wide-angle glaucoma, and who had been maintained on either 0.075- or 0.1-percent DFP for at least six months without any loss of visual field and with satisfactory diurnal intraocular pressure curves. These cases also were tried on 0.01-percent DFP.

All cases were followed for at least six months, and some for 12 months.

Results of study

The results can be analyzed by breaking these cases into several groups. The mild cases were divided arbitrarily into those that maintained daily intraocular diurnal curves ranging below 25 mm. Hg (Schiøtz) and those that had diurnal curves ranging above 25 mm. Hg while on pilocarpine. Of the 40 cases, eight fell in the latter group.

Variations of a comparable nature are found represented in Figure 1. The diurnal intraocular-pressure curves in the 32 eyes with simple glaucoma, which had been maintained on 0.5- to 1.0-percent pilocarpine nitrate three or four times daily, revealed an average fluctuation of intraocular pressure for every 24-hour period of 4.0 mm. Hg.

Instillation of DFP, 0.01 percent, either nightly or night and morning produced an intraocular pressure that averaged 5.0 mm. Hg lower than the pressure maintained with pilocarpine, and the average fluctuations in intraocular pressure throughout any 24-hour period was always less than 2.0 mm. Hg. In 14 eyes, the daily pressure plotting was practically a horizontal line while on 0.01-percent DFP.

In all eyes of this group treated with 0.01-percent DFP, at the end of six months it was noticed that the base intraocular pressure was at least 3.0 mm. Hg and in one eye 6.0 mm. Hg higher than that initially observed at the beginning of DFP therapy. However, the diurnal fluctuations were still not greater than 3.0 mm. Hg.

The rise in the base pressure was a gradual one during the six-month period. In eight of the eyes it was necessary to increase the frequency of instillation of 0.01-percent DFP from once to twice daily and, in four eyes, three times daily in order to maintain the pressure below 28 mm. Hg (Schiøtz).

In the eight eyes which had an intraocular-pressure curve ranging above 25 mm. Hg at the beginning of DFP therapy, all had been carried previously on either 2.0- or 4.0-percent pilocarpine instilled either four or five times daily. The fluctuations in the intradiem curves averaged 6.0 mm. Hg and, in one eye, the spread between the base pressure and the highest pressure was 8.0 mm. Hg (Schiøtz).

In these eyes the instillation of 0.01-percent DFP, night and morning (in two eyes at night, first thing in the morning, and during the afternoon), maintained the intraocular pressure at an average of 4.0 mm. Hg lower than that observed with pilocar-

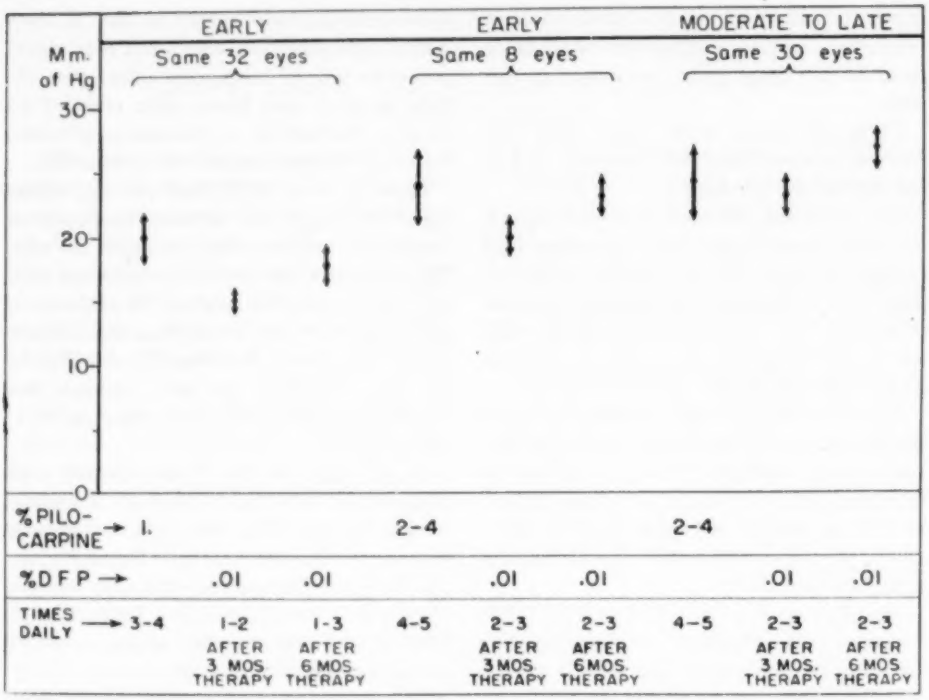


Fig. 1 (Leopold and Cleveland). A comparison of the effect of pilocarpine and of dilute DFP on the average of diurnal fluctuations in intraocular pressure.

pine therapy. The diurnal fluctuation was never greater than 3.0 mm. Hg in any of these eyes while on DFP instillations. At the end of six months, the base pressure had risen 2.0 to 6.0 mm. Hg. At the end of this time the diurnal fluctuations were still less than 3.0 mm. Hg.

In three eyes, at the end of six months, 0.035-percent DFP had to be used three times daily in order to maintain the pressure well below 28 mm. Hg to 30 mm. Hg.

All of the 30 eyes in the group with moderate to advanced glaucomatous field changes showed some changes in the anterior segment, such as iris atrophy, ectropion uveae, varying degrees of lens changes, and pigment on the posterior corneal surface. There were changes in the optic nervehead, consisting of cupping, varying degrees of pallor, and circumpapillary atrophy.

The intraocular pressure had been maintained below 30 mm. Hg (Schiøtz) by 2.0- to 4.0-percent pilocarpine instilled four to five times daily. On three months or more of this therapy, none of these eyes had shown any loss of visual field; 0.01-percent DFP therapy was used either two or three times daily in this group.

Prior to DFP therapy, diurnal fluctuations showed an average range of 5.0 mm. Hg. With DFP, the intradiem curve varied less than 3.0 mm. Hg. In this concentration, DFP carried the intraocular pressure at the same level or a few millimeters lower than that obtained with 2.0- or 4.0-percent pilocarpine nitrate solution.

At the end of six months, however, the base intraocular pressure had risen to as much as 3.0- to 4.0-mm. Hg in 24 of these eyes. The 24-hour intradiem pressure curves

showed a fluctuation of less than 3.0 mm. Hg even at the end of a six-month or longer period of observation.

In the 24 eyes that showed a gradual rise in base pressure not one showed any loss of visual field. The intraocular pressure of all could be reduced by increasing the strength of DFP to 0.035- or 0.05-percent, two or three times daily.

Of the eight eyes which had been controlled with 0.075- or 0.1-percent DFP and were switched to 0.01-percent DFP, all showed a rise of intraocular pressure on the lower concentrations of DFP. All these cases, prior to institution of DFP therapy, had failed to be controlled with pilocarpine in various concentrations and frequency, or 10-percent furmethide. The diurnal fluctuation on 0.075-percent DFP was less than 3.0 mm. Hg in any 24-hour period. On 0.01-percent DFP, the rise in intraocular pressure was as much as 8.0 mm. Hg in a majority of eyes and, in one, the pressure rose 12 mm. Hg. Instillations of 0.01-percent DFP were also made twice a day in this group. Increasing the frequency of instillation of 0.01-percent DFP decreased the pressure closer to its former level, but did not succeed in lowering the pressure to the former base-pressure.

Further comparisons of the data obtained in this study are summarized in Figure 2. This shows the strength of previous miotics used, the number of daily instillations required, and the tension ranges noted while on 0.01-percent DFP.

Several interesting observations can be made from this study. In most eyes in the group of medically controlled, chronic, wide-angle glaucoma, 0.01-percent DFP brought the intraocular pressure to a level lower than that maintained by pilocarpine nitrate in varying concentrations. Instillations of DFP did not have to be made as frequently as pilocarpine nitrate in order to maintain the pressure at this lower level.

Fluctuations in the diurnal curves were definitely less with the use of DFP than with pilocarpine nitrate. There was a gradual rise of the base pressure as the patient continued on DFP therapy, indicating either a change in the glaucomatous process in the eye or a decreasing response of the eye to DFP therapy for other reasons.

In all patients, with few exceptions, who were carried on pilocarpine nitrate and switched to 0.01-percent DFP, there was a definite increase in symptoms of ciliary spasm. For the first few days, and often few weeks, the patient complained of inability to accommodate to near and far vision, of blurred vision, and of browache and headache.

These symptoms definitely reduced in

NO. OF EYES	STAGE OF GLAUCOMA	PREVIOUS MIOTIC	DAILY INSTILL-ATIONS	TENSION RANGE ON PREVIOUS MIOTIC	DFP .01% DAILY FREQUENCY	TENSION RANGE ON DFP
		Pilocarpine		(mm. Hg)		(mm. Hg)
32	EARLY	1%	3-4x	15-25	1-2x	10-22
8	EARLY	2-4%	4-5x	19-30	2-3x	15-25
30	MODERATE TO LATE	2-4%	4-5x	19-30	2-3x	19-30
8	MODERATE	DFP .075-0.1	1-2x	18-28	3x	25-38

Fig. 2 (Leopold and Cleveland). Effect of dilute DFP (0.01 percent) in chronic simple wide-angle glaucoma.

severity as the patient continued on the drug. Some patients, however, had to be persuaded to continue with this miotic. Those who used 0.01-percent nightly stated that their vision became clear by noon or evening of the next day.

In one particular group of 22 eyes, 16 accepted the use of this concentration of DFP without unusual comment. Four of these patients requested that the drug be discontinued. However, they were persuaded to continue and after 10 days' use of the drug, tolerance was developed which made them receptive to its use. In the remaining two eyes, a very severe allergic reaction took place which was controlled with considerable difficulty. In these latter two eyes, the drug had to be discontinued.

There were at least three patients who had been controlled by dilute DFP but who had sudden rises in tension which could not be reconciled with our other findings. However, when more careful scrutiny was made of their supplies of miotic, it was discovered that in each case the drug (in peanut-oil) had been contaminated either by tears or by water on the dropper. It was found that, with any water contamination, this concentration of DFP quickly reduced its potency and control of the intraocular pressure was lost.

In this series of wide-angle glaucomas, no precipitous rise in intraocular pressure followed the use of fresh 0.01-percent DFP; nor were any retinal detachments noted.

Disadvantages of DFP

There are several disadvantages of this form of therapy:

1. Ciliary-spasm symptoms are greater than with pilocarpine but are usually tolerated, particularly after continued use. However, the ciliary-spasm symptoms will not be bothersome if the patient is first controlled by a strong miotic—for example, 0.075-percent DFP or 4.0-percent pilocarpine, and then placed on 0.01-percent DFP.

2. Care must be used by the patient to insure against contamination of the preparation with water for, in the dilute concentration, potency disappears rapidly on the slightest contact with water.

3. Sensitivity reactions may occur but are infrequent—only two occurred in the 78 eyes (both in same patient).

4. Apparently a tolerance or resistance to the effectiveness of 0.01-percent DFP may occur within six months of therapy. Stronger concentrations will be effective in some of these eyes.

Conclusions

1. DFP, 0.01 percent, controlled the intraocular pressure of eyes with mild, chronic wide-angle glaucoma.

2. The diurnal fluctuations in these eyes are less with 0.01-percent DFP than with pilocarpine nitrate therapy.

3. Fewer instillations of DFP 0.01-percent, were required than with pilocarpine nitrate.

4. DFP, 0.01 percent, produced mild but definite ciliary-spasm symptoms. These disappeared with continued use—they were not noticed if stronger concentrations of DFP had been used previously (for example 0.07 percent) and were less bothersome after previous use of 4.0-percent pilocarpine nitrate solution.

1930 Chestnut Street (3).

References

Banysek, J.: Di-isopropyl fluorophosphate: A new miotic. J. Ceskoslov. oft., **5**:8, 1949.

Ehlers, H.: Di-isopropyl fluorophosphate in the treatment of glaucoma. J. Ugesk. f. Leger, **112**:82, 1950.

Gallino, J. A., and Bottini, E. E.: Clinical observations on the use of DFP. Arch. Oftal, Buenos Aires, **24**:195, 1949.

Goedbloed, J.: Observations with di-isopropyl fluorophosphate. Ann. d'ocul., **182**:885, 1949.

Lebensohn, J. E.: di-isopropyl fluorophosphate (DFP) in the treatment of glaucoma. Arch. Ophth., **30**:1423, 1948.

Leopold, I. H., and Comroe, J. H., Jr.: Effect of di-isopropyl fluorophosphate (DFP) on the normal eye. Arch. Ophth., **36**:17, 1946; Use of di-isopropyl fluorophosphate (DFP) in treatment of glaucoma. Arch. Ophth., **36**:1, 1946.

Leopold, I. H., and McDonald, P. R.: Di-isopropyl fluorophosphate in the treatment of glaucoma. Arch. Ophth., **30**:1423, 1948.

Marr, W. G.: Clinical use of DFP in chronic glaucoma. Arch. Ophth., **30**:1423, 1947.

McDonald, P. R.: Treatment of glaucoma with di-isopropyl fluorophosphate (DFP). Am. J. Ophth., **29**:1071, 1946.

Raiford, M. B.: Clinical evaluation of DFP in glaucoma therapy. Am. J. Ophth., **32**:1399, 1949.

Scheie, H. G.: Glaucoma: A review of the literature, 1949-1950. Arch. Ophth., **44**:883, 1950.

Stone, W. C.: Use of di-isopropyl fluorophosphate (DFP) in the treatment of glaucoma. Arch. Ophth., **43**:36, 1950.

Weekers, R., and Rousel, F.: Experimental and clinical studies of the mode of action of retrobulbar injection of alcohol. Ophthalmologica, **118**:115, 1949.

Wheeler, J. R.: The reduction of the intraocular pressure by curare, retrobulbar procaine, and di-isopropyl fluorophosphate. Tr. Ophth. Soc. U. Kingdom, **69**:209, 1949.

OCULAR PEMPHIGUS

Report of a case with the histologic findings in the cornea

Peter S. Soudakoff,* M.D., and Harold F. Whalman,† M.D.
Los Angeles, California

Ocular pemphigus has been studied thoroughly both clinically and pathologically. The whole subject was reviewed by Klauder and Cowan[1] in 1942.

It is a well-known fact that in advanced cases the cornea is also involved and loses its transparency. While pathologic changes of the conjunctiva have been extensively studied, only five corneas have been examined pathologically.

Baeumler[2] attended a nine-year-old girl affected by pemphigus. She had extensive eruption over the body with involvement of both eyes. She developed a deep corneal ulcer in one eye. The anterior part of the eyeball was removed and the content of the eyeball was eviscerated.

The study of the removed portion of the eye revealed that, at the place of the ulcer, two thirds of the corneal stroma were destroyed. The corneal epithelium was thickened; the superficial cells were flattened, the middle ones were polyhedral and the basal were cylindrical. In several places a thin connective-tissue band was seen stretching between the epithelium and the stroma. Part of the corneal stroma was infiltrated with inflammatory cells and vascularized.

In Adam's case,[3] the eyeball was obtained at autopsy from a woman, aged 57 years, who had pemphigus bullae on the buccal mucosa and over the right arm. Both eyes were involved in the process.

Microscopic examination showed that the corneal epithelium had assumed an aspect of epidermis with numerous ingrowths. In places, Bowman's membrane was destroyed by degenerative pannus. The outer third of the corneal stroma was infiltrated with lymphocytes. The bulbar conjunctiva was transformed into cicatricial tissue which encroached upon the upper segment of the cornea in the form of a pterygium extending beneath the epithelium.

D'Amico[4] enucleated, because of perforating corneal ulcer, an eyeball affected by pemphigus. The corneal epithelium had the appearance of a pavement epithelium. In places where the bullae had ruptured, the epithelium was absent. Regeneration of the epithelium was handicapped by chronic in-

* From the Estelle Doheny Eye Foundation.
† From the College of Medical Evangelists.

flammation of the tissue lying beneath it. The epithelium, being edematous, had a tendency to become separated from the corneal stroma. This patient had also cutaneous pemphigus.

Smith and others[5] observed a woman, aged 61 years, both of whose eyes were affected by pemphigus. She also had eruptions of the buccal mucosa, over the right eyelids, and down the cheek. The right eye was enucleated because of a corneal ulcer and painful bullae.

Microscopic examination showed that the bulbar conjunctiva, considerably thickened near the limbus, had invaded the cornea like a pterygium and advanced between the epithelium and Bowman's membrane. The epithelium showed numerous ingrowths and, in a few places, was elevated to form small vesicles. The substantia propria was diffusely infiltrated with polymorphonuclear cells, especially around the ulcer which had destroyed the superficial lamellae. Capillaries invaded the deep layers of the cornea.

Kapuscinski[6] transplanted a cornea to an eye of a man, aged 60 years, who developed pemphigus of the eye and skin. The removed cornea was totally denuded of epithelium. Bowman's membrane was replaced by a membrane, hyaline in aspect, in which degenerated collagen fibers were detected. A granular mass lay upon the stroma. It was vascularized and infiltrated with lymphocytes at the limbus. Beneath the degenerative pannus degenerated collagen fibers were present.

Case report

A white woman, aged 77 years, was seen in the eye clinic of the White Memorial Hospital in February, 1951. She stated that she had had ocular pemphigus for 10 years.

On clinical examination it was found that the lower cul-de-sac of her right eye was completely obliterated. Pterygiumlike fleshy tissue, coming in from the limbus, covered the entire cornea. A crater-shaped ulcer was present in the lower segment at the 6-o'clock position. The corneal stroma beneath the ulcer was semitransparent. The fleshy growth could easily be peeled from the cornea.

The lower cul-de-sac of the left eye was completely obliterated by scars and adhesions. The cornea was transparent except for a triangular opaque area at the 7-o'clock position. This area had the appearance of sclerosing keratitis.

Vision was: R.E., light perception; L.E., 20/25.

Pemphigus bullae were present on buccal mucosa.

In view of the progressive corneal ulcer, the right eye was enucleated and submitted for microscopic examination.

Macroscopic examination. The cornea has been covered almost entirely by a white membrane growing in from the limbus. It has been peeled away from the 10- to 2-o'clock positions. The resulting flap of tissue is connected near the limbus from the 2:30- to 5-o'clock positions. Temporally the overlying opaque membrane is still adherent to the corneal surface from the 6- to 10-o'clock positions.

Beneath, the cornea is transparent; the iris and pupil can be clearly seen. The cornea is compressed inferiorly and a round white opacity one mm. in diameter is present just within the limbus at the 6-o'clock position.

On opening the eyeball, broad anterior synechias were found inferiorly.

Microscopic examination. The corneal epithelium is almost totally detached, only a small portion at the limbus remains adherent. The superficial cells are flattened and the outermost are keratinized; those in the middle are polyhedral, separated from each other, revealing the presence of intercellular bridges. Some of the cells have two nuclei and a great many contain two nucleoli. The basal cells are elongated.

The epithelium is about three to four times normal size due to edema and papillalike ingrowths. The epithelium rests upon loose connective tissue which is vascularized

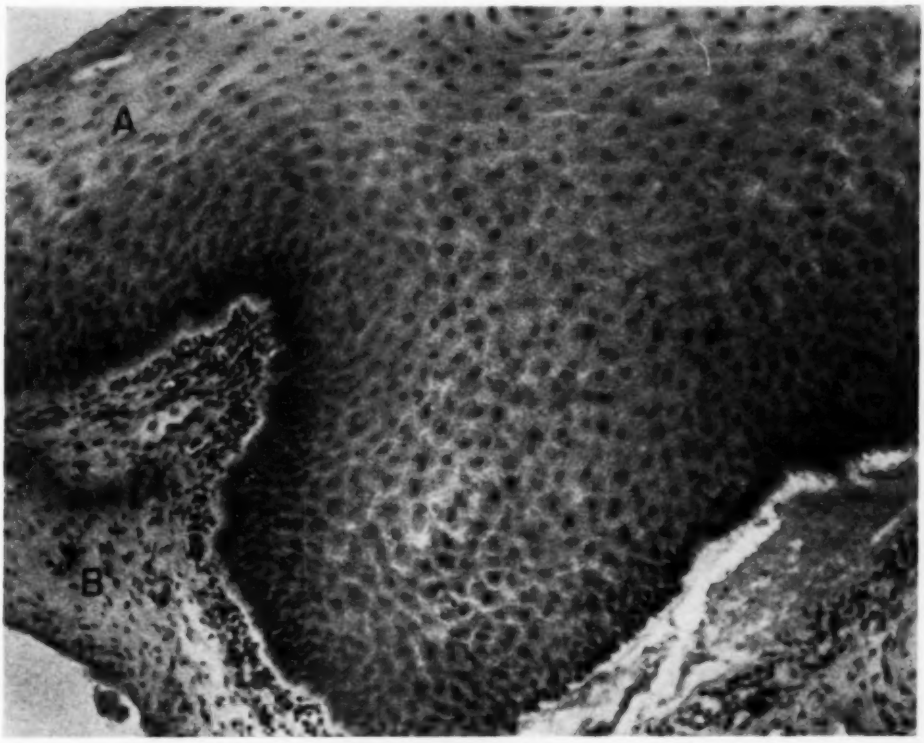


Fig. 1 (Soudakoff and Whalman). (A) Edematous corneal epithelium.
(B) Connective tissue beneath the epithelium.

and contains numerous round and elongated cells (fig. 1). Within the epithelium there are areas resembling those found in bullous keratitis.

Near the limbus two thirds of the corneal stroma have been destroyed. The walls of the ulcer show no infiltration with inflammatory cells. More centrally, Bowman's membrane has been eroded by the invading subepithelial membrane (fig. 2). Descemet's membrane and the endothelium are present.

The bulbar conjunctiva encroaches upon the cornea, and, like a degenerative pannus, it extends between the epithelium and Bowman's membrane.* This tissue is infiltrated with lymphocytes and polymorphonuclear leukocytes, and is richly vascularized (fig. 3).

There are very few keratic precipitates. Inflammatory cells are found upon and within the trabecular meshwork. One iris leaf shows ectropion uveae and the other moderate infiltration with polymorphonuclear leukocytes.

The lens shows minimal vacuolization at the equator within the cortex and nucleus. The posterior segment of the eyeball is not remarkable. There is marked vascularization and extensive infiltration of the perilimbal conjunctival tissue with lymph cells and plasma cells.

* It is difficult to state, at this point, whether the newly formed connective-tissue membrane in the cornea is a degenerative pannus or is an invasion of the newly formed connective-tissue membrane from the bulbar conjuctiva. Attempted differential staining in this case of pemphigus, as well as of known specimens of degenerative pannus, has so far failed to established a criterion for positive statement.

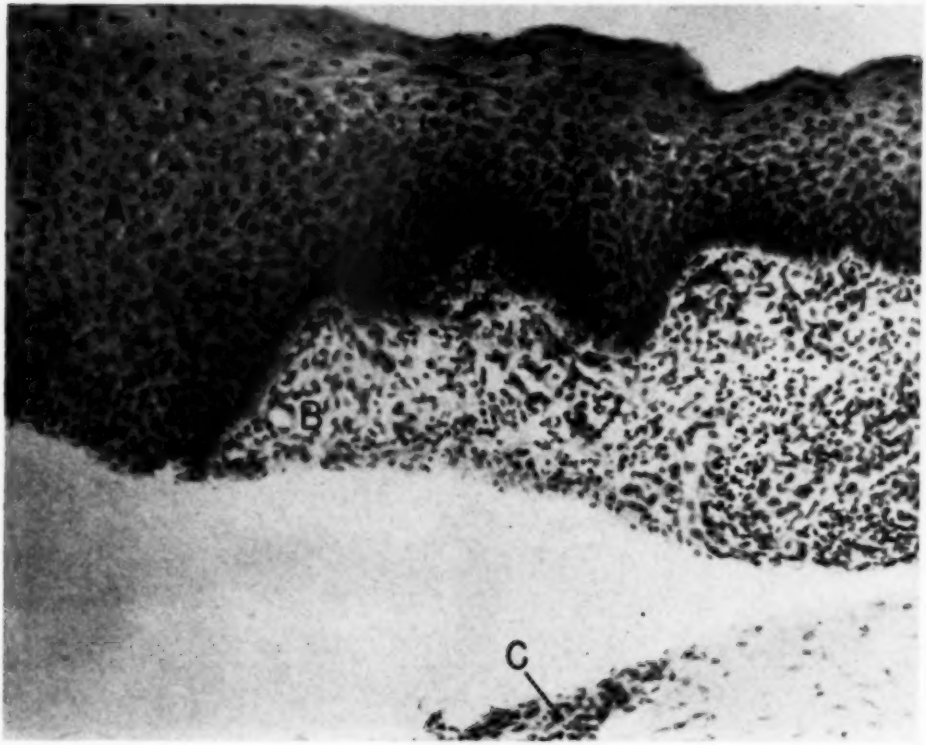


Fig. 2 (Soudakoff and Whalman). (A) Corneal epithelium. (B) Vascularized connective tissue extending between the epithelium and Bowman's membrane erodes corneal stroma at (C).

Comments

Comparing our findings with those of the previous investigators, we see that in all six cases the bulbar conjunctiva showed vascularization and infiltration with lymph cells and plasma cells. In cases complicated by a corneal ulcer polymorphonuclear leukocytes were also present.

In Adam's and Smith's cases, as well as in our case, the bulbar conjunctiva advanced upon the cornea, running between the epithelium and Bowman's membrane. In Adam's and Kapuscinski's cases, such tissue destroyed Bowman's membrane. In our case Bowman's membrane was destroyed by the corneal ulcer as well as at the place where the subepithelial membrane penetrated into the corneal stroma.

In two cases (Adam's and D'Amico's) the corneal epithelium assumed an epidermal appearance. In Smith's cases and in our case, the epithelium was edematous and showed bullae. Separation of the epithelium from Bowman's membrane was common to all six cases. Corneal ulcers were present in all cases except that of Adam.

In all cases of ocular pemphigus in which the cornea was examined microscopically, a vascularized membrane was found beneath the corneal epithelium and it was in direct contact with a similar membrane of the bulbar conjunctiva.

Villard[7] and his collaborators, who examined all cases of ocular pemphigus with the slitlamp, paid particular attention to the membranes arising from the limbus and spreading over the cornea. Very often the affected cornea had the appearance of

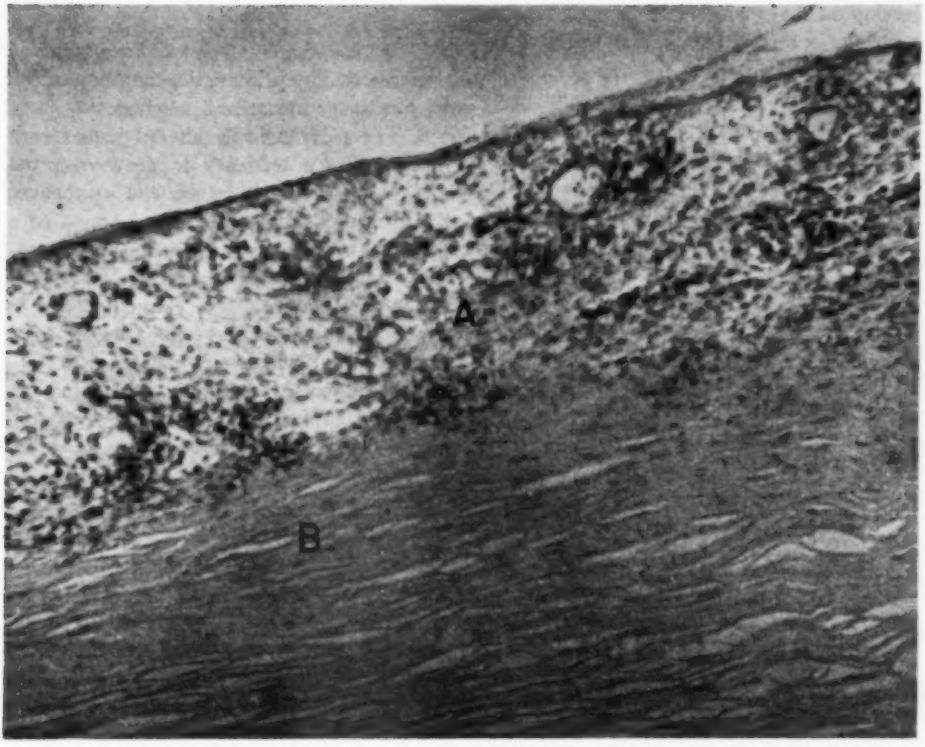


Fig. 3 (Soudakoff and Whalman). (A) Vascularized connective tissue of the bulbar conjunctiva extending into the cornea. (B) Corneal stroma.

sclerosing keratitis. Villard attached great importance to the presence of vessels in these membranes, but he could not decide whether the vessels were responsible for the process or whether they were sequelae of the process.

It is a well-established fact that conjunctivas affected by pemphigus contract, hence this disease has also been called essential shrinkage of the conjunctiva. Adam, Uhthoff,[8] D'Amico, and Sachsalber[9] attributed contraction of the conjunctiva to chronic inflammation.

In the cases of Dejean,[10] Sattler,[11] and Kapuscinski the conjunctivas were free from inflammatory cells. Because of this Kapuscinski thought that the principal cause of the pathologic changes was a primary degeneration of the tissues of ectodermic origin, while the inflammation was secondary to it.

The vascular membrane found beneath the corneal epithelium in cases of ocular pemphigus, being of the same structure as that in the bulbar submucosa, is also subject to cicatrization and this process has been found in several instances.

Diminution of vision in a case of corneal involvement is partially due to vascularization and partially to formation of a subepithelial membrane. Transformation of the epithelium into an epidermislike structure also may be a cause of diminished vision. Villard observed a parchmentlike appearance of the cornea.

Bullae develop quite frequently in far-advanced cases of ocular pemphigus. As Cogan[12] demonstrated, the vesicles and bullae

develop on the cornea when osmotic tension is greater in the epithelium than in the tears. This may happen when the salt content of the tears is decreased or the salt content of the cornea is increased.

In the advanced stage of pemphigus, the lacrimal puncta are often obliterated, the condition which reverses osmotic tension. At first, edema of the epithelium occurs; later, vesicles and bullae appear. Kapuscinski studied a case of pemphigus by daily examination of the cornea with a slitlamp. He noted that fully developed bullae disappeared on the following day, while new ones developed elsewhere.

In order to restore vision, peeling of the cornea was tried. This treatment improved the vision for only a short time (Kapuscinski). A corneal transplant failed to restore the vision because it turned opaque very soon. It, therefore, seems to us that, until the process in the bulbar conjunctiva is eliminated, the vascularized membrane will tend to grow into the cornea and any surgical procedure will produce only a temporary effect.

Atrophy and shrinkage of the conjunctiva cause diminution in the secretion of the meibomian and other secreting glands which lubricate the cornea. This results in desiccation of the cornea with consequent diminution of the vision. It is the second obstacle which must be surmounted before vision can be restored.

Summary

1. Ocular pemphigus is a disease which is characterized by primary degeneration of the conjunctival mucosa and submucosa.

2. The pathologically altered bulbar conjunctiva has a tendency to grow over the cornea, spreading between the epithelium and Bowman's membrane.

3. Diminished osmotic tension in the corneal epithelium, caused by lack of tears, predisposes to edema of the epithelium and to its separation from the stroma in the form of vesicles and bullae.

4. A vascular membrane developing beneath the corneal epithelium shows a tendency to cicatrization.

5. This membrane may erode Bowman's membrane.

6. Pathologic changes in the cornea are responsible for reduced vision. All surgical procedures attempted have failed to restore the vision, apparently because of the tendency of the newly formed connective-tissue membrane in the bulbar conjunctiva to grow into the cornea.

7. Atrophy and shrinkage of the conjunctiva diminish the secretion of meibomian and other secreting glands, which predisposes the cornea to desiccation.

2131 West Third Street (5).

References

1. Klauder, J. V., and Cowan, A.: Ocular pemphigus. Am. J. Ophth., **25**:643-661, 1942.
2. Baeumler, E.: Pemphigus Conjunctivae und essentialle Schrumfung. Klin. Monatsbl. f. Augenh., **23**:331-345, 1885.
3. Adam, C.: Untersuchungen zur Pathologie des Pemphigus Conjunctivae. Ztschr. f. Augenh., **23/1**: 48, 1910.
4. D'Amico, D.: Sulla istogenesi del pemphigo conjunctivale. Ann. ottal. e clin. Ocul., **53**:11-125, 1925.
5. Smith, E. C., Myers, E. A., and Lamb, H. D.: Ocular and oral pemphigus. Arch. Ophth., **11**: 635-640, 1934.
6. Kapuscinski, W. J.: Sur le pemphigus oculaire. Ann. d'ocul., **174**:451-473, 1937.
7. Villard, H., Dejean, C., and Dupponois, J. Examen biomicroscopique de deux cas de pemphigus oculaire. Bull. soc. d'opht. de Paris, **6**:358-363, 1930.
8. Uhthoff: Quoted by Kapuscinski.[6]
9. Sachsalber: Quoted by Kapuscinski.[6]
10. Dejean: Quoted by Kapuscinski.[6]
11. Sattler: Quoted by Kapuscinski.[6]
12. Cogan, D. G., Bullous keratitis. Arch. Ophth., **25**:941-968, 1941.

THE SURGICAL TREATMENT OF OCULAR PEMPHIGUS

M. Wallace Friedman, M.D.
Los Angeles, California
AND
Edwin S. Wright,* M.D.
North Hollywood, California

Pemphigus is a rare dermatologic disease of undetermined origin characterized by mucous membrane and cutaneous bullae and associated with submucous cicatrization. In ocular pemphigus the important pathologic process is the invasion of the submucosal tissue by newly formed connective tissue.[1] Bullae are, as a rule, not a discernible part of this disease entity.

There is a constantly increasing cicatricial contraction of the conjunctiva which progresses slowly, it is true, but is generally conceded to be irresistible.

Symptomatically the disease represents a picture of a chronic catarrhal conjunctivitis. Superimposed secondary infection may occur. Remissions and exacerbations are the rule, with the damage of each subsequent attack apparently added to that of its predecessor.

At first a thick, glary discharge is present. The serous exudation occurs submucosally, but the action of the lids causes the would-be bullae to break, leaving raw areas of conjunctiva. The conjunctiva grows more and more dry as the lacrimal secretions dry up, owing to the occlusion of the excretory ducts of the lacrimal gland by the shrinking conjunctiva. The raw spots become covered with a gray scarred coating, but may adhere to an opposing raw spot to form a symblepharon.[2]

The cornea may develop pannus and epithelial erosions and is further injured by both the loss of tear formation with its subsequent drying and the entropion with secondary trichiasis which follows cicatricial contraction.

The terminal stages are similar to xerosis with a parchmentlike appearance of the entire conjunctiva, which may be thrown into folds concentric to the cornea (already vascularized with emissary capillaries), with fine capillaries coming to the surface.[3] The end result of the uncontrolled case of ocular pemphigus may well be scarring of sufficient density to cause total blindness. This may be complicated by chronic corneal ulcers which keep breaking down.

The differential diagnosis in the earlier stages is from trachoma: (1) Pemphigus always shows a low-grade eosinophilia; (2) the cicatrization in trachoma involves the upper lid mostly; whereas, in pemphigus, the reverse is true and there may indeed be complete obliteration of the lower fornix with ankyloblepharon; (3) the pannus of pemphigus is always irregular as opposed to the symmetrical pannus of trachoma.[4]

Pathologically, the process is new formation of connective tissue in bulk with a small round-cell infiltration in and beneath the mucous membrane. Mucosa and submucosa are densely infiltrated, the infiltration being diffuse and nodular. The so-called "essential shrinkage of the conjunctiva" is due to the disappearance of the rich vascular network in the connective tissue with resultant cicatrization of the tissue.[5]

Treatment of this disease has always been unsatisfactory in the past. Medical treatment has been tried and relegated to the treatment of symptoms. Arsenic in full doses, quinine, salicylates, and other drugs have been tried without much success.[6] Whole-blood transfusions, intravenous mercurochrome, tartar emetic, and intramuscular turpentine in olive oil have been advocated.[7] Bland oils, antibiotics, and artificial tears are needed for dryness and secondary invaders. Local ultra-

* Assistant clinical professor of ophthalmology, College of Medical Evangelists, Los Angeles.

Fig. 1 (Friedman and Wright). The left eye with a skin transplant in the lower cul-de-sac, marked symblepharon, and limitation of motion. Hairs in the skin irritated the cornea.

violet, surface radium, and X rays have all been tried without specific result.[8]

Surgically, the epilation of the in-turning lids has been given precedence. Tarsorrhaphy has been attempted to alleviate the symptoms in later stages and to try to prevent corneal complications,[9] but more extensive procedures, such as wide excision and transplantation of mucous membranes, have been regarded as useless in checking the remorseless contraction of the underlying scar tissue toward obliteration of the sac.[10-13] Only Fuchs advised transplantation of pieces from another mucous membrane into the conjunctival sac in order to replace the conjunctiva that has been destroyed.[2]

Several cases of ocular pemphigus have recently been seen to progress to tragic conclusions. The case about to be presented represents an attempt surgically to control, or alleviate, the pathologic onslaught so as to prevent the hitherto inevitable ocular morbidity.

Case report

A 60-year-old white woman came to the eye clinic of the Los Angeles County General Hospital complaining of burning, smarting, tearing, and frequent foreign-body sensation in each eye, as well as occasional diplopia. She gave the history of having had chronically inflamed eyes for eight years. Her daughter had been forced to epilate the lashes at frequent intervals. Twenty-six years ago the patient noted vague blisters on her hands and forearms which recurred each year when she used strong solutions for housecleaning. No history of actual cutaneous pemphigus was elicited.

The original examination revealed a marked symblepharon in each eye with a low-grade conjunctivitis, bilateral entropion of the lower lids, and a glistening white skin transplant in the inferior cul-de-sac of the left eye. There was a diffuse pannus in the right eye, none in the left. The remainder of the examination was negative, with the exception of a grade-one hypertensive retinopathy present on fundus examination.

A biopsy taken from the symblepharon area in the right eye was reported, "There is a marked increase in capillaries and lymphatics in the subepithelial area with occasional polys and eosinophils. In one place there is a separation of the epithelium from the underlying stroma. The lesion is diagnostic of ocular pemphigus." Schirmer's test showed the filter paper wet four mm. in five minutes in the right eye, three mm. in five minutes in the left eye.

Fig. 2 (Friedman and Wright). The left eye after a large mucous-membrane transplant from the mouth had replaced the skin graft and the symblepharon had been dissected.

A six-mm., full-thickness, mucous-membrane graft into the right lower lid from a donor area in the mouth was done. Postoperatively the patient required two rows of Ziegler cautery punctures to combat entropion, otherwise the transplant was quite successful.

Six months after the transplant, the patient was admitted for a trial course of ACTH because of increased symblepharon and drying effect on the left eye. A full therapeutic dosage of intravenous ACTH was given with no effect. Entropion required frequent epilation.

Seven months after the original transplant, the patient was readmitted because of the development of an almost complete ankyloblepharon of the left eye with the added irritation of the hairs present in the skin graft of the lower left lid (fig. 1). A seven-mm., mucous membrane transplant into the lower lid of the left eye was done after the skin had been removed and the symblepharon freed. Again the mouth was the donor site. Healing took place without incident (fig. 2).

Fig. 3 (Friedman and Wright). Eighteen months after surgery with the patient looking straight ahead.

Fig. 4 (Friedman and Wright). Final result, patient looking to her left. Note that motion is not limited.

Fig. 5 (Friedman and Wright). Final result, patient looking to her right. Note that motion is not limited.

The patient has been followed in the eye clinic of the Los Angeles County General Hospital. When she was last seen, 18 months after the original surgery, she had almost complete range of motion and no recurrence of symblepharons (figs. 3, 4, and 5). There was no chronic conjunctivitis, no entropion, and, for the first time, no need for epilation.

Discussion

This case represents the successful surgical control of a disease that has previously been thought to be refractory to all forms of treatment. The technique of the mucous-membrane transplantations was only remarkable in that the recipient site was prepared by taking off the scarred tissue deeply. This was done to avoid repetition of the submucosal scarring if possible. Plastic stints were used immediately postoperatively to help keep the transplant in place and prevent unwanted adhesions. Direct sutures with 6-0 silk (Ethicon) were used. Pressure dressings were applied to the operative site for three days, after which simple eyepads were used. The donor sites were never sutured and healed in four days.

Conclusion

In order to prevent the obliteration of the conjunctival cul-de-sac by the pathologic process accompanying ocular pemphigus we recommend early and wide mucous-membrane transplants.

743 South Berendo Street.
10747 Riverside Drive.

References

Berens, C.: The Eye and Its Disease. Philadelphia, Saunders, 1949, p. 390.
Berliner, M. L.: Biomicroscopy of the Eye. New York, Hoeber, 1943, p. 222.
Duane, A.: Fuchs' Textbook of Ophthalmology. Philadelphia, Lippincott, 1917, ed. 5, p. 203.
Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1938, v. 2, p. 1728.
Fischer: Klin. Monatsbl. f. Augenh., **27**:439, 1889.
Gelpke: Klin. Monatsbl. f. Augenh., **23**:191, 1885.
Gunn: Tr. Am. Ophth. Soc., **13**:30, 1893.
Noyes: Rep. N. Y. Eye & Ear Infirmary, 1897.
Nuper: Am. J. Ophth., **17**:639, 1934; **18**:363, 1935.
Rycroft: Brit. J. Ophth., **18**:571, 1934.
Sommers, I. G.: Histology and Histopathology of the Eye. New York, Grune & Stratton, 1949, p. 495.

THE PULFRICH STEREO-EFFECT PRODUCED BY MONOCULAR MAGNIFICATION WITHOUT REDUCING ILLUMINATION*

PAUL W. MILES, M.D.
Saint Louis, Missouri

Pulfrich,[1] 1922, found that a pendulum moving rhythmically in a frontal plane in binocular observation soon begins to describe an ellipse when a density filter or stenopeic slit reduces the illumination of one eye. The pendulum movement is clockwise with the filter before the left eye, and counterclockwise when it is before the right.

The Pulfrich effect has been explained by an assumed delay in sensation time in the shaded eye. Here, the pendulum image constantly lags behind the position as imaged by the other eye, producing alternating stereoscopic disparity as the pendulum changes direction.

Wagner[2] has added that the Pulfrich effect increased with fatigue, decreased in high illumination, but was not affected by the emotional state. Pieron[3] showed that filters equal in physical density to light, but unequal in color, caused different degrees of the Pulfrich effect. That produced by red was least, while that for blue was greatest.

Engel and Fischer[4] described a difference in size and distance of the target at the extremes of the trajectory, which was more pear-shaped than an ellipse. They found an increase of apparent movement with greater target distance, and confirmed Monje's work that the Pulfrich effect is not strictly correlated with stereoscopic acuity. They suggested that the difference in target size at the extremes of pendulum swing was due to variation of convergence innervation, as in the wallpaper phenomenon.

The wallpaper phenomenon is a classic, which may be elicited by crossing the eyes on some repeating pattern with vertical lines. Depending on the extent of convergence, the pattern appears nearer and much smaller. This micropsia with convergence was discussed at length by Miles.[5]

Engel and Fischer considered the possibility that the Pulfrich effect might develop from delay in the neuromotor system of the extraocular muscles instead of the sensory system leading to stereopsis. Delayed eye movement would produce much the same type of disparity as illustrated in Figure 1.

They pointed out the similarity of the Pulfrich effect to the Mach-Dvorak[6] phenomenon. In the latter, only rapid or jerky movements are effective stimuli, as in the case of a cat stalking a bird. In the Pulfrich phenomenon, the greatest sagittal movement

* From the Department of Ophthalmology, Washington University School of Medicine, and the Oscar Johnson Institute.

occurs at the limits of the pendulum swing, and is greater with increased target speed.

Lythgoe[7] stated that dark adaptation of one eye did not produce the Pulfrich phenomenon, but that light to one eye from a flash light or transparent but reflecting glass produced the effect of a density filter before the other eye. Like many others, he concluded that delayed sensation and stereopsis can only partially explain the effect.

Without mentioning the similarity to the Pulfrich effect, Ames,[8] in 1946, described an experiment in which a six-inch white card was placed on the end of a six-foot wand. With the near end of the wand held near the chin, the card on the other end was moved rhythmically before the eyes in horizontal pendulum fashion. When a magnifying lens (two-percent, axis 90° aniseikonia) was placed before the left eye, the card appeared to move in a pear-shaped trajectory (fig. 2). In moving toward the left, the card appeared to gain speed, size, and distance. In moving toward the right it appeared to lose speed, size, and approach nearer. Ames used the phenomenon as one of many examples of distortion of commonplace environment by monocular change of retinal image size. He made no attempt to explain it.

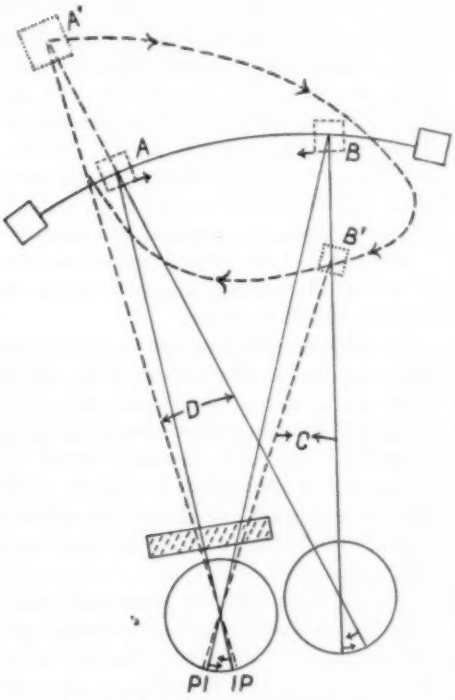


Fig. 1 (Miles). The Pulfrich effect.

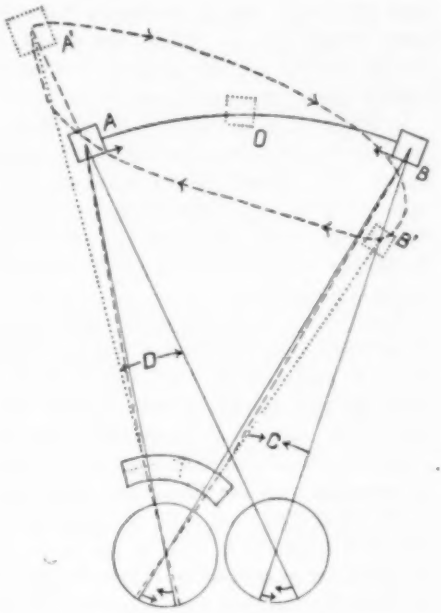


Fig. 2 (Miles). The Ames effect.

In Figure 1, the two eyes are shown as circles. The solid lines drawn through the rotation centers are near-foveal lines, indicating roving gaze on the square white card as it moves back and fourth. The slight lag in fixation of the right eye is not shown, but the greater lag of the retinal perception of the shaded eye is shown by dashed lines.

The dim optical image can be assumed to strike the retina at the solid line, but does not register until this portion of retina has moved to the position of the dashed line. Or, if the delay is in the motor system, one could assume the foveal line to lag behind in the

position of the dashed lines, while the optical image continued in the position of the solid lines.

However the means, the result is stereoscopic disparity as indicated in Figure 1, so that while the target is moving toward the right at A, it is seen in uncrossed disparity more remote at A′. While the target is moving toward the left at B, it is seen in crossed disparity more proximal at B′.

In Figure 2, as in Figure 1, the solid lines are near-foveal as the eyes follow the white square. The dashed lines indicate the prismatic effect of an afocal magnifier before the left eye, so that while the eye fixes A, or B, the line of direction in which it is perceived is A′ or B′.

Again, while A moves to the right, there is uncrossed disparity and a distant image. While B moves to the left, there is crossed disparity and an approaching image. The dotted lines are those projected from the displaced retinal image, and become the lines of direction used for fusion and stereopsis.

The chief difference from the condition in Figure 1 seems to be that in the center of the pendular movement 0, Figure 2, there is no disparity because the magnifier has no prismatic power in its optical center. In Figure 1, the density filter should produce disparity all across the field. If it does not, it is because of the Mach-Dvorak phenomenon of constant movement, and an inadequate stereoscopic stimulus.

The stereoscopic disparity illustrated in Figure 2 should be present with the target stationary. A difference in position and size is present if the background is dark, the targets bright, and the magnification more than three-percent. Linear, not massive targets, are best for detection of differences in image magnification (aniseikonia).

Both the Pulfrich and the Ames phenomena require several seconds of time. The trajectory, size, and distance changes appear identical. One might expect, therefore, that a density filter before the left eye would cancel the effect of a magnifier before the right eye. This is not the case, because the density filter always directs the movement.

The size changes of the target as it rotates should not be surprising. If a strong afterimage is impressed on one or both retinas, it appears to enlarge as the eyes fix a distant object, and shrink as they fix nearby. The size change in Figures 1 and 2 from A′ to B′ is about 30-percent, which is about the amount seen in the various experiments used to develop the horopter projection size theory.[5]

Letter C and its arrows in the figures suggest that in gaze to the right, there is innervation for convergence. This might well be, because disparity there is crossed. Letter D and its arrows suggest innervation for divergence in leftward gaze, with consequent effect in perceived size according to the projection on a distant horopter.

In the Pulfrich and Ames effects, there is an increased movement if the pendulum string or the wand is observed instead of the end target. The linear target support in each instance is a stereoscopic stimulus, being mostly vertical in position, and best in strong color contrast with the background. If the supporting string or wand is invisible, the effects are greatly reduced. In the Ames effect, one is almost convinced the wand is made of rubber, it stretches so freely.

It seems to make little difference whether or not head movements are permitted in the Pulfrich and Ames effects.

The changes in apparent speed of the target are due to the differences in sagittal movement in gaze to the right compared to the left. Where the space traversed is great, apparent speed must increase. When the space covered is small, as in looking to the right in Figures 1 and 2, the movement to consume the same amount of time must appear slowed.

The Pulfrich and the Ames effects are ordinarily reversible, in that the exact opposite movement occurs with transfer of the density filter or the magnifier to the other eye. This, however, is not true of individuals

with high ocular dominance. The filter or the magnifier may cause a recessive eye to suppress, which would reduce the stereo-effect with the device before the recessive eye.

It is unknown whether the Pulfrich and the Ames effect occur naturally in patients with anisocoria and anisometropia. Further studies are indicated from both clinical and theoretical points of view.

Summary and Conclusion

The Pulfrich and the Ames effects are subjectively exactly similar. Either a density filter or a magnifying lens may be placed before one eye and produce the elliptical movement of a pendulum swinging in the frontal plane. The similarities described indicate that stereopsis in each case is a very important factor.

The effective stimulus differs from most stereoscopic conditions, however. The apparent elliptical figure does not follow any other but reciprocal movement, and is most noticed at the points where acceleration or deceleration of the target is great.

640 South Kingshighway (10).

References

1. Pulfrich, C.: Die stereoskopie im Dienste der isochromen und heterochromen photometrie. Naturwiss, **10**:25, 553, 1922.
2. Wagner, E.: Observations on the Pulfrich effect. Klin. Monatsbl. f. Augenh., **115**:143-149, 1949.
3. Pieron, H.: Chromatic specificity in the latency of luminous responses. Rev. Optic. Th. Instr., **28**:157-160, 1949.
4. Engel, H. J., and Fischer, M. H.: Optokinetic perception of space, a new concept. Pflug. Arch. ges. Physiol., **253**:1-27, 1950.
5. Miles, P. W.: The relation of perceived size of half images at the fusion level to projection on the horopter. Am. J. Ophth., **34**:1543-1561, 1951.
6. Mach, E., and Dvorak, V.: Sitzb. kgl. bohm. Ges. Wiss., **65**:1872. Quoted by Duke-Elder, W. S.: Textbook of Ophthalmology, Mosby, St. Louis, 1939, v. 1, p. 629.
7. Lythgoe, R. J.: Some observations on the rotating pendulum. Nature, (London), **141**(1):474, 1938.
8. Ames, A., Jr.: Binocular vision as affected by relations between uniocular stimulus patterns in commonplace environments, Am. J. Psychol., **59**:333-357, 1946.

KIMMELSTIEL-WILSON SYNDROME*

Review of the literature and report of a case with the ophthalmoscopic findings

Martin Cohen, M.D.
New York

A case of diabetic retinopathy with associated kidney symptoms is described in this paper. This combination of ocular and renal symptoms is sometimes called the Kimmelstiel-Wilson syndrome, since the renal lesion usually present in such cases was first described by these authors. A brief review of reports relating to the renal lesion follows.

Kimmelstiel and Wilson,[1] in 1936, described a focal hyalinization in the renal intercapillary connective tissue of the glomerular tuft in patients with diabetes mellitus. This lesion was termed "intercapillary glomerulosclerosis" and they associated it with a clinical syndrome which they claimed was almost as characteristic as the histologic lesion. The diagnosis of intercapillary glomerulosclerosis was made by them on the basis of necropsy findings in seven diabetic patients.

Kimmelstiel and Porter,[2] in 1948, discussing the differential diagnosis between chronic glomerulonephritis and intercapillary glomerulosclerosis, stated that they were two distinct, although very similar, diseases.

* Read before the New York Society for Clinical Ophthalmology, January 7, 1952.

They also maintained that the retinopathy was directly dependent on the diabetes rather than on the renal lesion; also that the nodular type of intercapillary glomerulosclerosis occurred in approximately 17 percent of diabetics and the nephrotic syndrome in less than 10 percent of patients presenting intercapillary glomerulosclerosis at autopsy.

Siegal and Allen,[3] in 1941, considered the clinical and pathologic features of this subject and reported only one case of intercapillary glomerulosclerosis in a series of 100 consecutive patients with nondiabetic hypertension.

Allen,[4] in 1941, reported that the Kimmelstiel-Wilson syndrome was found in 33 percent of diabetics over the age of 40 years. He regarded intercapillary glomerulosclerosis as a focal intramural glomerulosclerosis (that is, arising within the capillary walls) rather than an intercapillary condition. He maintained that the hyalinized laminated nodules in this lesion could be considered the most reliable criterion for the renal pathologic diagnosis of diabetes mellitus in patients over 40 years of age.

Rifkin[5] and his associates, in 1948, stated that intercapillary glomerulosclerosis was known to exist in diabetes; also that a distinct clinical picture was associated with so-called diabetic glomerulosclerosis, which includes glycosuria, edema, hypertension, combined diabetic and hypertensive retinopathy, albuminuria, and lipoid cells in urinary sediment.

Bell,[6] in 1942, described two types of glomerular lesions characteristic of diabetes —the nodular and the diffuse. He considered "nodular or spherical type" almost pathognomonic of diabetes.

Henderson, Sprague, and Wagener,[7] in 1947, found intercapillary glomerulosclerosis in 19.5 percent of 313 diabetics on whom necropsy was performed. They did not consider the lesion a specific one, at least morphologically, as shown by the fact that it was found in 12.3 percent of 81 cases of chronic glomerulonephritis and in 5.2 percent of 134 patients dying of hypertension and its complications.

Wagener believed retinopathy to be a common accompaniment of this condition, and stated that practically all cases of intercapillary glomerulosclerosis included some form of diabetic retinopathy; also that the incidence is more closely related to duration of diabetes than to the chronologic age of the patient.

Many additional reports pertaining to the renal phase of this condition have appeared in the literature. Some authors, including Horn and Smetana,[8] Laipply, Eitzen, and Dutra,[9] Goodof,[10] and others have stated that so-called diabetic glomerulosclerosis is also present in nondiabetics. Allen has stated that diabetic glomerulosclerosis is a lesion specific for diabetes mellitus if the rarest equivocal cases are expected.

Diabetes mellitus generally precedes intercapillary glomerulosclerosis by several years; the glycosuria affecting diabetics probably alters biochemically the circulation, causing sclerotic lesions in the renal and ocular vasculature.

Mann and others[11] found, in a series of diabetics, a higher incidence of intercapillary glomerulosclerosis in a considerably lower age group than is indicated in other reports.

Case report

History. C. S., a man, aged 52 years, was admitted to the medical clinic of the New York Post-Graduate Hospital in the spring of 1943. He had had diabetes mellitus, intermittently controlled by insulin and diet, for 17 years, and ankle edema, moderate systemic hypertension, and renal disease for one year. His vision had also been impaired for one year. A diagnosis of Kimmelstiel-Wilson syndrome was recorded. On September 21, 1943, he was admitted to Grasslands Hospital, his principal complaints again being ankle edema and impaired vision. There were also symptoms of cardiac decompensation. His past history was noncontributory.

Physical examination showed enlargement of the heart, enlarged liver, ankle

edema, and marked clubbing of the fingers. Blood pressure was 174 mm. Hg, systolic and 108 mm. Hg, diastolic.

Laboratory findings. Electrocardiogram showed myocardial damage and left ventricular hypertrophy. *Urinalysis:* Specific gravity, 1.012 to 1.018; albumin, 4 plus; occasional hyaline and granular casts.

Blood count: Hemoglobin, 12 gm. per 100 cc.; red blood cells, 4,250,000; white blood cells, 9,080 with 84 percent polymorphonuclear leukocytes. *Blood chemistry:* Nonprotein nitrogen increased from 75 mg. percent on admission to 167 mg. percent on discharge. *Fasting blood sugar:* Normal (under control). *Creatinine* increased from 2.0 mg. percent on admission to 4.6 mg. percent on discharge. *Total serum protein:* 6.0 gm. per 100 cc. and 6.2 gm. percent on two occasions. *CO_2 combining power:* 65 vol. percent. *Whole blood chlorides:* 500 mg. percent. *Mosenthal two-hour concentrating test:* Specific gravity varied from 1.011 to 1.015.

Clinical course. The cardiac decompensation improved somewhat following the usual supportive therapy. The patient was discharged at the end of four months with a discharge diagnosis of diabetes mellitus, hypertensive cardiovascular disease, and arteriolar nephrosclerosis. He died seven months later of uremia and cardiac failure. Autopsy permission was not obtainable.

Eye examination. Deterioration of vision was the only external symptom.

Funduscopic findings. Both fundi were similarly involved. The optic discs were sharply defined and slightly pale. The perimacular area contained many tiny, glistening, sharply circumscribed whitish foci. Five pinpoint hemorrhages were scattered over the equatorial area of the fundus. The retinal veins were moderately congested, while some arterioles showed slight uneven contractions. These fundus changes are characteristic of diabetic retinopathy.

Further changes were observed in the arterioles; their lumina had a silver-wire appearance, and some were contracted and uneven in caliber. In areas where they crossed retinal veins, arterioles caused nicking of the underlying vein. These vascular changes are typical of arteriolar sclerotic retinopathy.

Other alterations were three so-called "discrete woolly exudates" and two linear

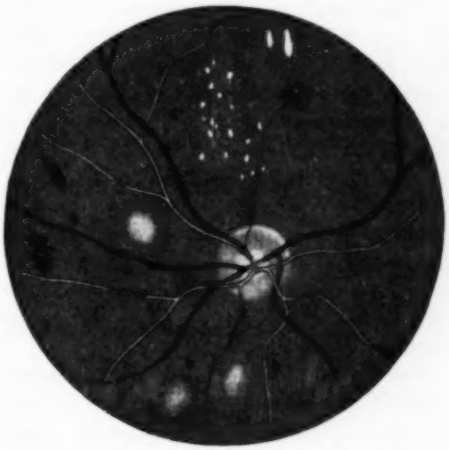


Fig. 1 (Cohen). Fundus findings in Kimmelstiel-Wilson syndrome. (Drawing from a Kodachrome film.)

whitish exudates situated in the equatorial area, together with two large striated flame-shaped hemorrhages above the disc in the periphery of the fundus. Such changes occur in patients with hypertensive retinopathy.

Whether these various fundus lesions are the result of primary systemic disease, especially diabetes mellitus, or whether they represent complicating lesions independent of the primary disturbance, is impossible to state, since their sequence has not been reported. There was no edema of the optic disc or retina, macular changes, or marked venous congestion, indicating that there was no severe renal lesion at the time of this ocular examination. The pinpoint retinal hemorrhages were completely absorbed after two weeks, ruling out microaneurysms in this case; later the woolly exudates also were absorbed. The retinal sclerosis was probably permanent. A fundus picture is shown in Figure 1.

The following publications refer to the ophthalmic aspect of this subject.

Cohen,[12] in 1929, stated that diabetic retinopathy is due mainly to vascular sclerosis of some retinal vessels, and that diabetes in time affects the fundus lesions, as in retinal and renal sclerosis.

Ballantyne,[13] in 1945, expressed the opinion that microaneurysms may occur alone and seem to be the earliest pathologic change in the fundus of diabetic patients. In a communication to the Ophthalmological Society of the United Kingdom, in 1943, Loewenstein and Ballantyne, referring to the so-called punctate hemorrhages of diabetic retinitis, expressed the belief that at least some of the small round hemorrhages situated at or near the perimacular vessels were actually microaneurysms as proven by further histologic study.

Friedenwald,[14] in 1948, referred to Kimmelstiel and Wilson's description of a form of "intercapillary glomerulosclerosis" which he believed was characteristic of the diabetic nephropathy. In the material gathered by him and his associates they found innumerable retinal capillary aneurysms in every case of obvious Kimmelstiel-Wilson disease that they studied. In this paper he strongly supported Ballantyne's conclusion that capillary aneurysms in the fundus are a characteristic feature of diabetic retinopathy and quite typically distinguish this lesion from the retinopathy of arteriosclerosis or of malignant hypertension.

White and Waskow,[15] in 1948, reported on 387 juvenile diabetics. They found that autopsy reports since 1940 on every diabetic child in the Joslin Clinic who had survived 15 years of diabetes showed the lesion of intercapillary glomerulosclerosis.

MacKenzie,[16] as early as 1876, requested Nettleship to make a complete microscopic examination and report of an enucleated eye of his diabetic patient. This report proved the existence of retinal aneurysms located on one side of the vessel wall.

In a group of cases described by Wexler and Branower[17] in 1950, numerous capillary lesions occurred in every case of proved malignant nephrosclerosis; their pathologic study of the retina revealed microaneurysms in every case of diabetes.

Ashton,[18] in 1949, found that intercapillary glomerulosclerosis in diabetes is regularly associated with retinal microaneurysms, whether distinguishable or not ophthalmoscopically. In a microscopic study of the tissues of 21 cases, including the eye structures, the brain, and several other organs, he found capillary microaneurysms solely in the retinal tissues.

Ashton,[19] in 1951 reported on microaneurysms in 250 nondiabetics. Of this number, one third of the cases showed microaneurysms after various operations and one third following eye diseases at post-mortem examination. The aneurysms were found in thrombotic glaucoma, chronic uveitis, glioma, and melanoma. He noted that there seemed to be no direct relation between the age of patients in this condition or their blood pressure. The kidney tissue was microscopically examined.

In my opinion, the diagnosis of retinal microaneurysms is usually verified by histologic examination of the retinal tissue; furthermore a few microaneurysms may be seen microscopically in normal retinal tissue.

Summary

1. Intercapillary glomerulosclerosis is a renal degenerative lesion prevalent in diabetes mellitus. The condition was first described by Kimmelstiel and Wilson in 1936.

2. The diagnosis of intercapillary glomerulosclerosis can be verified only by a postmortem renal examination, but a long-standing case of mild diabetes with retinopathy and renal dysfunction is strong evidence of a Kimmelstiel-Wilson lesion.

3. It has been stated that intercapillary glomerulosclerosis occurs in nondiabetics, but this contention in most of the reported cases has not been confirmed by Allen's investigations.

4. Retinal capillary microaneurysms have been observed and recognized in the early

stages of diabetes mellitus, but are considered by some authors nonspecific for diabetes.

5. The prognosis of intercapillary glomerulosclerosis, if clinically established, depends on the severity of the associated lesions, as well as upon the effect of recognized treatment for diabetes mellitus.

6. Continued investigation of this subject is important, in order to evaluate the varied opinions which have recently appeared in the literature.

29 East 64th Street (21).

References

1. Kimmelstiel, P., and Wilson, D.: Intercapillary lesions in the glomeruli of the kidney. Am. J. Path., **12**:83, 1936.
2. Kimmelstiel, P., and Porter, W. B.: Intercapillary glomerulosclerosis. New England J. Med., **238**:876, 1948.
3. Siegal, S., and Allen, A. C.: Intercapillary glomerulosclerosis (Kimmelstiel-Wilson) and the nephrotic syndrome in diabetes mellitus. Am. J. M. Sc., **201**:516, 1941.
4. Allen, A. C.: So-called intercapillary glomerulosclerosis: A lesion associated with diabetes mellitus. Arch. Path., **32**:33, 1941.
5. Rifkin, J., Parker, J. G., et al.: Diabetic glomerulosclerosis. Medicine, **27**:429, 1948.
6. Bell, E. T.: Renal lesions in diabetes mellitus. Am. J. Path., **18**:744, 1942.
7. Henderson, L. I., Sprague, R. C., and Wagener, H. P.: Intercapillary glomerulosclerosis. Am. J. Med., **3**:131, 1947.
8. Horn, R. C., and Smetana, H.: Intercapillary glomerulosclerosis. Am. J. Path., **18**:93, 1942.
9. Laipply, T. C., Eitzen, O., and Dutra, F. R.: Intercapillary glomerulosclerosis. Arch. Int. Med., **74**:354, 1944.
10. Goodof, I. I.: Intercapillary glomerulosclerosis. Am. Int. Med., **22**:373, 1945.
11. Mann, G. V., Gardner, C., and Root, H. F.: Clinical manifestations of intercapillary glomerulosclerosis in diabetes mellitus. Am. J. Med., **7**:3, 1949.
12. Cohen, M.: The eye in diabetes mellitus. Arch. Ophth., **2**:529, 1929.
13. Ballantyne, A. J.: Retinal changes associated with diabetes and with hypertension. Arch. Ophth., **33**:97, 1945.
14. Friedenwald, J. S.: A new approach to some problems of retinal vascular disease. Tr. Am. Acad. Ophth., **73**: (Nov.-Dec.) 1948.
15. White, P., and Waskow, E.: Arteriosclerosis in childhood diabetes. Proc. Am. Diabetes A., **8**:139, 1948.
16. MacKenzie, S.: A case of glycosuric retinitis, with comments. Ophthalmic Hosp. Rep., London, **9**:pt. 1, 134, 1876.
17. Wexler, D., and Branower, G.: Retinal capillary lesions in malignant hypertension. Arch. Ophth., **44**:539, 1950.
18. Ashton, N.: Vascular changes in diabetes, with particular reference to the retinal vessels. Brit. J. Ophth., **33**:407, 1949.
19. ———: Retinal micro-aneurysms in the non-diabetic subject. Brit. J. Ophth., **35**:189, 1951.

THE INFLUENCE OF CORTISONE ON THE RATE OF BLOOD ABSORPTION FROM THE EYE

WALTER H. BENEDICT,* M.D., AND ROBERT W. HOLLENHORST,† M.D.
Rochester, Minnesota

In a previous study in which cortisone was used and found to have no effect on fibroblastic proliferation in dogs' eyes, it was noted incidentally that in hyphemic eyes, the blood was absorbed more slowly in those eyes in which cortisone was used.[1] Consequently, since a search of the recent literature could not uncover any specific information on this side effect, it was decided to pursue this observation further.

Blunt, Plotz, Lattes, Howes, Meyer, and Ragan[2] however, when describing the pathologic changes in fractures in rabbit femurs

* Fellow in Ophthalmology, Mayo Foundation, University of Minnesota.
† Section of Ophthalmology, Mayo Clinic.

The following publications refer to the ophthalmic aspect of this subject.

Cohen,[12] in 1929, stated that diabetic retinopathy is due mainly to vascular sclerosis of some retinal vessels, and that diabetes in time affects the fundus lesions, as in retinal and renal sclerosis.

Ballantyne,[13] in 1945, expressed the opinion that microaneurysms may occur alone and seem to be the earliest pathologic change in the fundus of diabetic patients. In a communication to the Ophthalmological Society of the United Kingdom, in 1943, Loewenstein and Ballantyne, referring to the so-called punctate hemorrhages of diabetic retinitis, expressed the belief that at least some of the small round hemorrhages situated at or near the perimacular vessels were actually microaneurysms as proven by further histologic study.

Friedenwald,[14] in 1948, referred to Kimmelstiel and Wilson's description of a form of "intercapillary glomerulosclerosis" which he believed was characteristic of the diabetic nephropathy. In the material gathered by him and his associates they found innumerable retinal capillary aneurysms in every case of obvious Kimmelstiel-Wilson disease that they studied. In this paper he strongly supported Ballantyne's conclusion that capillary aneurysms in the fundus are a characteristic feature of diabetic retinopathy and quite typically distinguish this lesion from the retinopathy of arteriosclerosis or of malignant hypertension.

White and Waskow,[15] in 1948, reported on 387 juvenile diabetics. They found that autopsy reports since 1940 on every diabetic child in the Joslin Clinic who had survived 15 years of diabetes showed the lesion of intercapillary glomerulosclerosis.

MacKenzie,[16] as early as 1876, requested Nettleship to make a complete microscopic examination and report of an enucleated eye of his diabetic patient. This report proved the existence of retinal aneurysms located on one side of the vessel wall.

In a group of cases described by Wexler and Branower[17] in 1950, numerous capillary lesions occurred in every case of proved malignant nephrosclerosis; their pathologic study of the retina revealed microaneurysms in every case of diabetes.

Ashton,[18] in 1949, found that intercapillary glomerulosclerosis in diabetes is regularly associated with retinal microaneurysms, whether distinguishable or not ophthalmoscopically. In a microscopic study of the tissues of 21 cases, including the eye structures, the brain, and several other organs, he found capillary microaneurysms solely in the retinal tissues.

Ashton,[19] in 1951 reported on microaneurysms in 250 nondiabetics. Of this number, one third of the cases showed microaneurysms after various operations and one third following eye diseases at post-mortem examination. The aneurysms were found in thrombotic glaucoma, chronic uveitis, glioma, and melanoma. He noted that there seemed to be no direct relation between the age of patients in this condition or their blood pressure. The kidney tissue was microscopically examined.

In my opinion, the diagnosis of retinal microaneurysms is usually verified by histologic examination of the retinal tissue; furthermore a few microaneurysms may be seen microscopically in normal retinal tissue.

Summary

1. Intercapillary glomerulosclerosis is a renal degenerative lesion prevalent in diabetes mellitus. The condition was first described by Kimmelstiel and Wilson in 1936.

2. The diagnosis of intercapillary glomerulosclerosis can be verified only by a postmortem renal examination, but a long-standing case of mild diabetes with retinopathy and renal dysfunction is strong evidence of a Kimmelstiel-Wilson lesion.

3. It has been stated that intercapillary glomerulosclerosis occurs in nondiabetics, but this contention in most of the reported cases has not been confirmed by Allen's investigations.

4. Retinal capillary microaneurysms have been observed and recognized in the early

stages of diabetes mellitus, but are considered by some authors nonspecific for diabetes.

5. The prognosis of intercapillary glomerulosclerosis, if clinically established, depends on the severity of the associated lesions, as well as upon the effect of recognized treatment for diabetes mellitus.

6. Continued investigation of this subject is important, in order to evaluate the varied opinions which have recently appeared in the literature.

29 East 64th Street (21).

References

1. Kimmelstiel, P., and Wilson, D.: Intercapillary lesions in the glomeruli of the kidney. Am. J. Path., **12**:83, 1936.
2. Kimmelstiel, P., and Porter, W. B.: Intercapillary glomerulosclerosis. New England J. Med., **238**:876, 1948.
3. Siegal, S., and Allen, A. C.: Intercapillary glomerulosclerosis (Kimmelstiel-Wilson) and the nephrotic syndrome in diabetes mellitus. Am. J. M. Sc., **201**:516, 1941.
4. Allen, A. C.: So-called intercapillary glomerulosclerosis: A lesion associated with diabetes mellitus. Arch. Path., **32**:33, 1941.
5. Rifkin, J., Parker, J. G., et al.: Diabetic glomerulosclerosis. Medicine, **27**:429, 1948.
6. Bell, E. T.: Renal lesions in diabetes mellitus. Am. J. Path., **18**:744, 1942.
7. Henderson, L. I., Sprague, R. C., and Wagener, H. P.: Intercapillary glomerulosclerosis. Am. J. Med., **3**:131, 1947.
8. Horn, R. C., and Smetana, H.: Intercapillary glomerulosclerosis. Am. J. Path., **18**:93, 1942.
9. Laipply, T. C., Eitzen, O., and Dutra, F. R.: Intercapillary glomerulosclerosis. Arch. Int. Med., **74**:354, 1944.
10. Goodof, I. I.: Intercapillary glomerulosclerosis. Am. Int. Med., **22**:373, 1945.
11. Mann, G. V., Gardner, C., and Root, H. F.: Clinical manifestations of intercapillary glomerulosclerosis in diabetes mellitus. Am. J. Med., **7**:3, 1949.
12. Cohen, M.: The eye in diabetes mellitus. Arch. Ophth., **2**:529, 1929.
13. Ballantyne, A. J.: Retinal changes associated with diabetes and with hypertension. Arch. Ophth., **33**:97, 1945.
14. Friedenwald, J. S.: A new approach to some problems of retinal vascular disease. Tr. Am. Acad. Ophth., **73**: (Nov.-Dec.) 1948.
15. White, P., and Waskow, E.: Arteriosclerosis in childhood diabetes. Proc. Am. Diabetes A., **8**:139, 1948.
16. MacKenzie, S.: A case of glycosuric retinitis, with comments. Ophthalmic Hosp. Rep., London, **9**:pt. 1, 134, 1876.
17. Wexler, D., and Branower, G.: Retinal capillary lesions in malignant hypertension. Arch. Ophth., **44**:539, 1950.
18. Ashton, N.: Vascular changes in diabetes, with particular reference to the retinal vessels. Brit. J. Ophth., **33**:407, 1949.
19. ———: Retinal micro-aneurysms in the non-diabetic subject. Brit. J. Ophth., **35**:189, 1951.

THE INFLUENCE OF CORTISONE ON THE RATE OF BLOOD ABSORPTION FROM THE EYE

WALTER H. BENEDICT,* M.D., AND ROBERT W. HOLLENHORST,† M.D.
Rochester, Minnesota

In a previous study in which cortisone was used and found to have no effect on fibroblastic proliferation in dogs' eyes, it was noted incidentally that in hyphemic eyes, the blood was absorbed more slowly in those eyes in which cortisone was used.[1] Consequently, since a search of the recent literature could not uncover any specific information on this side effect, it was decided to pursue this observation further.

Blunt, Plotz, Lattes, Howes, Meyer, and Ragan[2] however, when describing the pathologic changes in fractures in rabbit femurs

* Fellow in Ophthalmology, Mayo Foundation, University of Minnesota.
† Section of Ophthalmology, Mayo Clinic.

treated with adrenocorticotropic hormone (ACTH) and cortisone, briefly mentioned that muscle hematomas in rabbits which received these hormones were still "jelly-like" at the end of two days, while organization and absorption of the hematomas in the untreated controls were already well under way after the same time interval.

The poor response to the use of cortisone and ACTH in some forms of senile macular degeneration, in the disciform type of macular degeneration, and in inflammatory types of choroidal or retinal disease in which there is a definite hemorrhagic component, might be partly explained if the hormones actually retard the rate of absorption of the hemorrhage. The same is also true in intraocular hemorrhage resulting from any cause.

Since recurrent ecchymosis is a part of the disease complex in Cushing's syndrome, in which a hyperactivity of the suprarenal gland exists, there may be some direct relationship between elevated levels of the 17-ketosteroids and the production of hemorrhages. If this were so, the use of cortisone would be contraindicated in diseases or conditions in which hemorrhage plays a prominent part.

For absorption to take place from tissues, new blood vessels must be formed and invading fibroblasts and histiocytes must break up and carry off the debris. If these elements do not make their appearance or if they are reduced in number, the process is absent or markedly delayed. That cortisone and ACTH reduce the formation of new blood vessels and granulation tissue and decrease capillary permeability is well known. Therefore, the rapidity with which blood is absorbed may be inversely proportional to the degree of hyperadrenalism.

The absorption of blood from the anterior chamber is of course not accompanied by the ingrowth of new blood vessels and scar tissue except under the most unusual circumstances. On the other hand, an increased capillary permeability of the ocular vessels probably does play a large part in the role of absorption of blood. Therefore, if such an important phenomenon is blocked by hormone therapy, the mechanism for resolving the hyphema will become altered. As regards the vitreous hemorrhages, the same situation no doubt exists although on a somewhat modified basis.

That the use of cortisone is actually contraindicated in eyes with intraocular hemorrhage is shown by the following experiment.

Experimental procedure

Eight rabbits received an injection of 0.2 cc. of their own venous blood (obtained from the marginal vein of the ear) into the anterior chamber of their respective right eyes. After cocainization of the globe, an oblique puncture was made through the cornea with a No.-27 intradermal needle attached to a tuberculin syringe, the blood injected, and the needle withdrawn. Although, in every case, a little aqueous humor seeped out through the puncture wound after removal of the needle, it was not thought to be an appreciable amount.

In each case, the intraocular pressure immediately became markedly elevated so that corneal edema was apparent. In some instances, the edema disappeared shortly after the needle was withdrawn but in others it persisted throughout the experiment. No doubt, the difference in behavior of the tension was attributable to the amount of aqueous humor lost immediately after withdrawal of the needle. Four of the rabbits were then left alone as controls. They received no further medication or treatment of any kind. The other four animals were given a subconjunctival injection of six mg. of cortisone every day, but no other form of medication.

Results

In each case, it was evident that the rate of absorption of blood in the cortisone-treated animals was delayed as compared to the rate of absorption in the untreated

animals. In some, there was a marked difference, whereas in others the time difference was not as striking, but in all the findings were consistent.

During the 27 days of observation, the hyphema in two of the untreated eyes was completely absorbed in 15 and 19 days respectively. Two of the cortisone-treated animals died from an overdose of the anesthetic agent (pentobarbital sodium) on the 15th day after injection of the drug. In these animals, the hyphema still occupied from a fifth to a fourth of the anterior chamber, while at the same time, in the untreated animals, the hyphema occupied considerably less volume.

When comparable rates of absorption of blood in all of the treated and in all the untreated animals were averaged, it was found that approximately the same amount of blood was absorbed about six and a half days faster from the eyes of the untreated animals than from the eyes of the animals treated with cortisone.

The cortisone did not appear to have any effect other than to retard the rate of absorption of blood. In the cortisone-treated animals, after all or most of the blood had been absorbed, the eyes appeared to have suffered no harm, except for the traumatic condition of the conjunctiva. In the eyes of these animals, the conjunctiva was congested, and, later on in the course of the experiment, it became somewhat scarred and adherent to the sclera because of repeated subconjunctival injections.

In one pair of rabbits whose anterior chambers received an injection of blood as described, glaucoma developed and the corneal stroma became markedly hazy. However, by the 16th day after injection, the cornea of the control animal was practically crystal clear while that of the cortisone-treated animal remained hazy except for very slight clearing.

The clearing of the cornea in the control animal was preceded by the development of a pannus which eventually extended two to three mm. onto the corneal surface. This pannus, which became apparent on the sixth day after injection, grew and persisted for eight days. In six to eight days it receded, with marked and rapid clearing of the cornea accompanied by a return of the intraocular pressure to normal.

A pannus never developed in the eye of the cortisone-treated rabbit. The cornea remained hazy, and the intraocular pressure remained elevated throughout the course of the experiment.

There were individual variations among the control and among the cortisone-treated animals as to the rate of absorption of blood, but, when individual control animals were compared to individual treated animals, the differences were still constant and significant, animal for animal.

Conclusions

Cortisone injected subconjunctivally in hyphemic eyes retards the rate of absorption of the blood. Consequently, it is emphasized that the use of cortisone is definitely contraindicated in the treatment of hyphema and probably also in the treatment of other conditions associated with intraocular hemorrhage.

The Mayo Clinic.

References

1. Benedict, W. H.: The influence of cortisone on the trephine wound in the eye in experimental animals. Thesis, University of Minnesota Graduate School, 1952.

2. Blunt, J. W., Jr., Plotz, C. M., Lattes, R., Howes, E. L., Meyer, K., and Ragan, C.: Effect of cortisone on experimental fractures in the rabbit. Proc. Soc. Exper. Biol. & Med., **73**:678-681 (Apr.) 1950.

NYSTAGMUS*

C. Wilbur Rucker, M.D.
Rochester, Minnesota

Nystagmus may be a helpful diagnostic sign. It is true that knowledge of its neural basis is incomplete and that at times its interpretation is puzzling. Yet, by arranging what is known about it in an orderly fashion it is possible to dispel some of the confusion and to make clinical use of the presence of nystagmus.

Nystagmus may be defined as an involuntary to-and-fro movement of the eyes. It apparently results from a disturbance within a complex apparatus, designed to keep the eyes in constant relation to their environment, which is intimately concerned with equilibrium. This apparatus consists of the visual system, the labyrinths of the inner ears, the extraocular muscles and portions of the central nervous system.

Lesions affecting some parts of this apparatus produce to-and-fro movements of the eyes that differ from those movements produced by lesions in other parts. Consequently, from the nature of the nystagmus, one can sometimes determine the site of the lesion responsible for it.

Nystagmus may be classified in a variety of ways.[1-8] No method is entirely satisfactory, and any presented at this time must be tentative. For clinical purposes certain advantages obtain in an anatomic classification such as that presented in Table 1.

Those varieties of nystagmus produced by disturbed vision are placed in Group I, those due to stimulation or depression of the labyrinths in Group II, those caused by weakness of the extraocular muscles in Group III, and those arising in the central nervous system in Group IV. In every classification of nystagmus there are a few varieties that do not seem to fit, and for these a miscellaneous group is required which is here termed Group V.

Optic nystagmus

This group includes the types originating within the visual system. There are two varieties, one that is physiologic and another that results from defective vision. They are altogether dissimilar in cause and in appearance.

Optokinetic nystagmus

This type, called also "railway" nystagmus, is induced by continuous movement of points of fixation past the eyes, as occurs in watching a moving train of cars or in looking out the window of a moving car. It may be elicited by rotating a striped drum before the eyes.

In fixing successive points of interest, the eyes repeatedly follow one object out of range and then jump back to pick a new point. The to-and-fro movement of the eyes thus attains a slow and a quick component, a pattern that resembles nystagmus originating in several other ways.

TABLE 1
Classification of nystagmus

I.	Optic
	A. Optokinetic
	B. Defective vision
II.	Labyrinthine
	A. Rotational
	B. Caloric
	C. Peripheral vestibular disease
	D. Postural vertigo
III.	Muscular
	A. Physiologic
	B. Paretic
IV.	Central
	A. Cerebellum
	B. Region of vestibular nucleus
	C. Medial longitudinal fasciculus
V.	Miscellaneous
	A. Hereditary
	B. Latent
	C. Occupational
	D. Voluntary and hysterical
	E. Toxic

* From the Section of Ophthalmology, Mayo Clinic. Read at the meeting of the Pacific Coast Oto-Ophthalmological Society, Salt Lake City, Utah, April 27 to May 1, 1952.

The neural mechanism for the pursuit type of nystagmus is complex. Impulses arising in the retinas are carried back through the visual pathway to the visual cortex on the medial surface of the occipital lobes.

Surrounding the visual cortex is an area, connected by association fibers with the visual cortex of its own side and by colossal fibers with that of the opposite side, from which impulses pass forward through the internal sagittal stratum, down the posterior limb of the internal capsule and lateral part of the cerebral peduncle, to the nuclei for eye movements in the midbrain and pons.

Changes in the pursuit pattern caused by lesions affecting this arc have been reported,[9] but they appear to possess only limited clinical application.

Nystagmus caused by defective vision

This represents a searching movement of the eyes, seemingly made in a vain effort to find a point of fixation.[10] Normally, the desire for macular fixation and the ability to maintain it develop during the first few months after birth. Should this desire be frustrated, the searching movement may continue and persist throughout life.

Searching nystagmus occurs in almost all cases of bilateral congenital defects of the macula, in cases of corneal scars from ophthalmia neonatorum, and in about half the cases of congenital cataract. In albinism, aniridia, and in complete color blindness, the nystagmus seems to be due to the presence of small central scotomas.

The nystagmus due to defective vision is rapid and pendular, lacking quick and slow phases; it is present on forward gaze and may increase in amplitude on gaze to the sides; it is most often horizontal and it persists during the entire lifetime of the patient.

It seldom occurs in persons who have lost macular vision after the first few months of life, nor does it occur in the completely blind. The eyes of the blind wander in a slow irregular fashion over a wide range, but not in a pattern that could be called "nystagmus."

Labyrinthine nystagmus

This group contains the varieties of nystagmus which arise as a result of disturbance in the peripheral vestibular apparatus of the inner ear, either from stimulation or from depression. Those varieties induced by the clinician may be regarded as physiologic; those occurring as a result of disease, pathologic. All are rhythmic, possess quick and slow components, and are accompanied by distressing vertigo.

For diagnostic purposes otologists study the eye movements after turning the patient in a Bárány chair and after irrigating the external auditory canal with warm or cold water.

Rotational nystagmus

Rotation of the head produces nystagmus in the plane of rotation, regardless of the position of the head. It seems to be due to inertia of the fluid within the semicircular canals.

At the beginning of turning there is a lag of the endolymph; when turning suddenly stops the momentum of the endolymph tends to keep it moving. The eyes are drawn in the direction of flow of endolymph. This represents the slow phase of the nystagmus. The quick, compensatory phase is in the opposite direction. Because the flow of endolymph relative to the canals is reversed when turning is suddenly stopped, the nystagmus also reverses its direction then.

Clinically, only the nystagmus persisting after the turning has ceased is studied. Its slow component is in the direction of turning and its quick component is in the opposite direction. Its amplitude is increased when the patient looks in the direction of the quick phase and is diminished when the patient looks in the direction of the slow phase. Change of gaze, although it alters the intensity of the nystagmus, does not alter the direction of its quick and slow components.

Caloric nystagmus

Stimulation of the vestibule by application of heat or cold also brings about a pro-

nounced nystagmus. Flushing an auricular canal with cold water causes a rhythmic movement of the eyes with the quick component to the opposite side; warm water in the canal produces a rhythmic movement with the quick component to the same side.

This phenomenon has not been explained to the satisfaction of all investigators. Bárány,[11] who performed much of the original work on this problem, stated that a thermal current is induced in the endolymph. If it is assumed that cooled endolymph falls and that warmed endolymph rises, then the nystagmus produced by this method is similar to that induced by turning. Acceptance of this hypothesis, however, is not universal.[12]

Nystagmus from peripheral vestibular disease

Irritative lesions of the labyrinth give rise to mixed horizontal and rotary nystagmus with the quick component beating to the side of the lesion. It is always of short duration, for, unless the lesion heals quickly, it becomes destructive, and when it does, the nystagmus reverses its direction.

A destructive lesion of the vestibule or vestibular nerve has a depressive effect, causing deviation of the head and eyes toward the side of the lesion. The slow phase of the nystagmus likewise is toward the side of the lesion, while the quick phase is to the opposite side. As in rotational and caloric nystagmus, turning the eyes toward the quick phase increases its amplitude, but change of gaze will not alter the direction of the quick and slow components.

When a vestibular nerve is severed, vertigo persists for one to several weeks. Because most lesions of the eighth nerve involve cochlear fibers as well as vestibular, there is an accompanying loss of hearing. If the disease process is chronic, deafness may be the only symptom.

Nystagmus is present during the episodes of dizziness in Ménière's disease. It is also a horizontal and rotary type of nystagmus, but the side toward which it beats is not significant.[13]

When impairment (either partial or complete) of the vestibular nerves is bilateral and equal, there is no nystagmus and no vertigo even in the acute phase.[14]

Postural vertigo

This is a symptom of a disorder in which there appears to be hyperirritability of the labyrinth to movement of the head. Some writers on the subject consider the basic lesion to be in the otolith apparatus of the labyrinth, while others suggest the possibility of a vascular disturbance in the brain stem.

The vertigo occurs in brief attacks induced by sudden changes in position of the head and is accompanied by nystagmus of a mixed rotary and vertical type. Often the first attack is the most intense; the successive attacks, recurrent over a period of a few weeks or months, gradually become less distressing and finally disappear.

The cause of the disorder is not known. It is generally a benign condition, for only seldom is it a symptom of demonstrable disease of the central nervous system. It does not affect the results of the usual functional tests of the labyrinth.[15]

In the diagnosis of postural vertigo, a helpful maneuver is to have the patient sit on the end of an examining table, turn his head toward one shoulder, and lie back quickly. The maneuver is repeated with the head turned toward the other shoulder. If postural vertigo is present, vertigo and nystagmus are induced by one or the other of these maneuvers, almost never by both. It is with the head turned toward the right that the symptoms arise in most cases, according to my experience.

After the patient lies back, there is a latent period of about two seconds before the onset of the vertigo and nystagmus, which in turn last only a few seconds. If the maneuver is repeated a few times, a refractory phase becomes established and during the next hour or more change in posture will no longer induce vertigo or nystagmus.

It is helpful for the patient to learn that

he can arise and lie down without becoming dizzy if he will move slowly and keep his head turned toward the side on which vertigo is not produced.

More elaborate maneuvers for studying the nystagmus elicited by changes in posture in patients with brain tumors are described by Nylén of Upsala.[16]

Muscular nystagmus

Physiologic neuromuscular nystagmus

This is also known as "end-position" nystagmus. It is elicited on extreme lateral gaze and consists of short jerks with the quick component in the direction of gaze. The jerks are usually greater in the outturning eye and may be present in it alone. These irregular movements occur in many normal individuals when they look beyond the limit of binocular fixation and they are of no special significance. Fatigue is a predisposing factor.

Paretic nystagmus

This sometimes accompanies palsy of extraocular muscles and is present in the field of action of the paretic muscles. Weakness of extraocular muscles or impairment of their motor nerves may render them incapable of holding the eyes steady when they are turned away from the midline. The quick component represents an intermittent attempt on the part of the muscle to do its work; the slow component is a temporary relaxation.

The nystagmus may be dissimilar in the two eyes or may be present in only one eye. In myasthenia gravis, nystagmoid movements are encountered occasionally when the eyes are turned into the field of action of the weakened muscles.

Partial paralysis of conjugate gaze also may give rise to the paretic type of nystagmus. In an effort to overcome the paralysis, intermittent jerking movements occur which, if they are repeated with sufficient frequency and regularity, may warrant the designation of nystagmus. The quick component is in the direction of gaze. This variety could be classified with nystagmus originating in the central nervous system.

Central nystagmus

Nystagmus also may originate as a result of lesions in the central nervous system. It does not arise from lesions in the basal nuclei or the cerebral hemispheres. It is produced only by lesions within the posterior fossa. Almost always, nerve bundles having connections with the vestibular nuclei are implicated.

The nystagmus of central origin may be horizontal, vertical, rotary, or dissociated. It is rhythmic, has a quick and a slow phase, and is of long duration, lasting for months or years. In contrast to the nystagmus of labyrinthine origin, vertigo is seldom associated with it. Other signs of intracranial involvement may be present to aid in the diagnosis.

Cerebellum

Although nystagmus is a frequent symptom of cerebellar disease, the role of the cerebellum in its production is uncertain, for usually there is associated involvement of the vestibular nerve or nucleus, or the roof nuclei of the cerebellum.[17] Characteristically the direction of the quick and slow movements is dependent on the position of the eyes and changes with change of direction of gaze. Two types of nystagmus are associated with cerebellar disease.

In one the eyes are steady on forward gaze but when the eyes are turned to the sides horizontal nystagmus is elicited, with the quick component in the direction of gaze and the slow component toward the midline. It resembles somewhat neuromuscular paretic nystagmus except that it is more intense and there is no associated paresis.

It is encountered in widespread destruction of the cerebellum, but is seen also in the presence of localized lesions. Clinically, it may be difficult to determine whether the lesion is in the cerebellum or in its afferent or efferent pathway.

In the other cerebellar type the point of rest is not in the midline but is located at an angle of from 10 to 30 degrees to one side.

As in the previous type, the slow phase is toward the rest point, the quick phase away from it. On forward gaze, there is a small rhythmic nystagmus, its quick phase beating away from the rest point. When the eyes are turned to the side, away from the rest point, the oscillations become slow and coarse. When the patient looks far to the opposite side, beyond the rest point, the excursions are rapid and small.

This variety of nystagmus was encountered frequently by Holmes[18] in his study of acute cerebellar injuries and was regarded by him as indicative of a destructive lesion on the side opposite the rest point.

Neurologists recognize that in a majority of instances the coarse beat of this type of nystagmus points to the side of the lesion, but there are so many exceptions that it is not a reliable lateralizing sign. This type of nystagmus may not be due directly to the cerebellar lesion, but to an effect on other structures, notably connections with the vestibular nucleus.

Region of the vestibular nucleus

Lesions affecting the vestibular nuclei or their connections with the cerebellum or brain stem may induce any one of several varieties of nystagmus, the particular form depending on the location of the lesion. Knowledge available at the present time does not permit the naming of specific pathways for specific types of nystagmus.

Vertical and rotary forms are characteristic. Lesions in this region also can cause forms of horizontal nystagmus similar to those which arise in the cerebellum. Mixed varieties are frequent.

Vertical nystagmus, beating with its quick component upward, increases in intensity on upward gaze, disappears on downward gaze, and is affected little or not at all when the eyes are turned to the sides.

Downward-beating nystagmus increases on gaze down or to the sides, and disappears when the eyes are turned upward in the direction of the slow component.

Most authors seem to have accepted the doctrine that vertical nystagmus is caused by a lesion in the superior part of the vestibular nucleus.[19] Tentatively this is acceptable for the upward-beating nystagmus. The site of origin of the downward-beating nystagmus is less definite, because evidence of cerebellar disease is usually associated with it.

Rotary nystagmus appears to be positive evidence of involvement of the vestibular nucleus.[20] It is described as clockwise or counterclockwise, according to the direction of the quick component. It often increases on lateral gaze, but does not change its direction on change of gaze; for example, a clockwise beat remains clockwise whether the eyes are turned to the right or to the left.

Rotary nystagmus is noted in occlusion of the posterior inferior cerebellar artery, and may occur in acoustic neuroma and in multiple sclerosis. In general, clockwise rotary nystagmus indicates a lesion on the right and is of greatest amplitude on gaze to the right; counterclockwise rotary nystagmus indicates a lesion on the left and is of greatest amplitude on gaze to the left. This generalization seems to hold true for infarctions within the brain stem, but with tumors there are so many exceptions that the direction of the nystagmus is not to be assigned lateralizing value unless it is consistent with other neurologic findings.

Medial longitudinal fasciculus

This is a bundle of association fibers which lies near the midline in the floor of the fourth ventricle and extends up through the midbrain and down through the medulla. It serves as a path for reflex control of the eyes, head, and neck, and also links together the oculomotor and vestibular nuclei. Most of the axones comprising it arise in the vestibular nuclei.

Interruption of its fibers or those of the neighboring reticulate substance disturbs the harmonious working-together of the two

eyes, and gives rise to dissociated types of nystagmus in which the movements of the eyes are unequal or dissimilar. These include internuclear ophthalmoplegia and monocular and see-saw nystagmus.

A lesion which involves the medial longitudinal fasciculus immediately above the level of the abducens nucleus causes internuclear ophthalmoplegia, a special variety of paresis of conjugate gaze, in which there is paralysis of the medial rectus muscle of the in-turning eye and coarse horizontal nystagmus in the out-turning eye.[21]

Most often this condition is bilateral and it is then almost pathognomonic of multiple sclerosis.[22] When it is unilateral it may be attributable to multiple sclerosis, but if the onset is sudden, it is more likely due to infarction.[23] Even in its complete bilateral form ability to converge is retained.

The sites of the lesions responsible for the other types of dissociated nystagmus, that is, the monocular[24] and see-saw[25] forms, have not been ascertained. A plausible possibility is that they are in the reticular formation of the upper part of the pons.

A small lesion within the posterior fossa can be depended on to produce the type of nystagmus specifically attributable to the particular location of such a small lesion. However, the neural elements in this area lie so close together and are so intermingled that large lesions, especially tumors, affect several structures simultaneously, and produce combinations of nystagmus that are almost unpredictable. It is for this reason that nystagmus has not been a reliable localizing sign for tumors within the posterior fossa.

Miscellaneous types of nystagmus

Hereditary nystagmus

This type appears as a constant, horizontal, pendular movement of the eyes, lacking quick and slow components. The excursions are small on forward gaze, become coarser on gaze to the sides, and beat at the rate of about 120 per minute.

Nystagmus in this condition resembles that of defective vision, yet visual acuity is usually good (generally 20/30 to 20/50) in spite of the constant motion of the eyes. Vision would probably be normal if the eyes could be made to stand still.

The nystagmus is usually first noticed by the mother when the child is a few weeks to a few months of age. It persists throughout life. In some instances there is an accompanying horizontal shaking of the head which is aggravated by attempts to see more clearly or by excitement.

The nature of the defect responsible for hereditary nystagmus is not known. It is inherited most frequently as a sex-linked character, in some pedigrees as recessive, in others as an irregular dominant, on the basis of a gene on the X chromosome. Manifestation is suppressed in one third to one half of the women carrying the gene.[26]

Latent nystagmus

This is a condition in which the eyes are steady when both are open but in which a bilateral horizontal nystagmus is induced when one or the other eye is occluded.

Its quick component is toward the side of the uncovered eye. There is no nystagmus when both eyes are closed or when both are occluded. Vision is good when both eyes are open, but there is considerable reduction in acuity when one is covered because of the resulting movement of the eyes.

There is no lesion in the retina and refractive error does not appear to be a factor. In most cases there is strabismus, either convergent or divergent.

The nystagmus is greater when the better of the two eyes is covered, and in some instances may be induced by covering one eye but not by covering the other.[27] It is present from infancy and remains unchanged. Reported cases lack a coördinated clinical picture and the theories proposed to explain the condition are widely discordant.[28] For all that, there is a recognized entity which is best termed latent nystagmus.

Occupational nystagmus

This type includes several varieties. They are all rare, and since I have not seen any of them myself, I shall simply mention two.

Miners' nystagmus,[29] formerly a common cause of industrial disability in Great Britain and on the continent of Europe, occurs among men who have worked underground in coal mines under poor illumination for 20 years or more. It is associated with tremor of the head, vertigo, and intolerance to light. The movements of the eyes are very rapid (about 150 or more per minute), are rotary in the form of a circle, are increased on upward gaze or on exertion, and are lessened when the person looks down. The patient prefers to walk with head thrown back and downcast eyes. The pathologic basis is not known. It is seldom seen in the United States.

Train dispatchers'[30] nystagmus has been reported. In this condition the movements of the eyes are horizontal and variable, and there is accompanying blepharospasm, visual impairment, and ocular discomfort.

Voluntary and hysterical nystagmus

These conditions are identical. A few persons have learned to produce nystagmoid movements of their eyes at will, either for the amusement of their friends or for use as a manifestation of conversion hysteria.

When the muscles in and about the orbits are suddenly tightened, the eyes are made to jerk rapidly in a clonic manner at a rate of several hundred times per minute. These movements occur through a small range and persist for periods lasting from five to 20 seconds. It has been described as a "shuddering" or "quivering" of the eyes.

The direction of these movements is largely horizontal with superimposed rotary elements, and the action may consist of a swift series of convergences. Since this type of nystagmus resembles none of the others it presents no diagnostic difficulties. Perhaps it should not even be called "nystagmus."

Toxic nystagmus

This may be induced by a wide variety of drugs, notably the opiates, barbiturates, and ethyl alcohol. In this type the eyes are steady on forward gaze; when they are turned to the sides or sometimes upward, nystagmus appears with its quick component in the direction of gaze. It resembles the type which is generally regarded as due to cerebellar deficit.

The site of action of the sedatives may not be on a single structure, for the barbiturates have been shown to act on the cerebral cortex, the brain stem, and intermediate neural pathways.[31] These same drugs abolish optokinetic, latent, positional, voluntary, and congenital nystagmus, and they alter or decrease nystagmus due to disease of the brain stem.

Clinical application

Examination for nystagmus may be conducted concurrently with the examination for a manifest weakness of the extraocular muscles. The eyes are observed both on forward gaze and as they are turned to the right and to the left. The patient is then asked to turn his eyes up and down while looking first to the right and then to the left. Finally, while he looks forward he is asked once again to look up and then down. The oblique positions do not yield as much information concerning nystagmus as do the movements to the sides and straight up and down.

When the patient looks straight ahead one of three possible conditions will prevail: (1) There may be a horizontal pendular movement of the eyes, (2) there may be a jerky movement in any direction, or (3) there may be no movement.

If the movements are pendular, they will increase in amplitude when the person looks to the sides and they may assume quick and slow components when the patient looks far to the sides.

For this type of nystagmus there are two possible causes. It may be the result of defective vision, and, if it is, the cause of the

reduction in vision should be apparent to the physician. If the eyes appear sound and if vision is not far from normal, the nystagmus is probably hereditary, and careful questioning regarding the family history will usually yield information concerning relatives who are similarly afflicted.

If, on forward gaze, there is a jerky nystagmus beating with quick and slow components, there are two possible sources; it may be of labyrinthine origin or it may arise in the central nervous system.

If it is labyrinthine, there will be an accompanying vertigo; if it is the result of a lesion in the central nervous system, vertigo is unlikely. In the absence of vertigo the physician determines if the direction of the nystagmus is horizontal, vertical, or rotary.

Horizontal nystagmus present on forward gaze will increase in amplitude when the eyes are turned in the direction of the quick component and will disappear when the eyes turn in the direction of the slow component. When the patient looks far in the direction of the slow component, beyond the rest point, nystagmus will again be elicited, this time in a direction opposite that present on forward gaze. This variety of nystagmus is of cerebellar origin, and the lesion is likely to be on the side toward which the quick component beats on forward gaze, and opposite to the side of the rest point.

If vertical nystagmus is present, it is usually visible on forward gaze. If it beats upward it increases on upward gaze, and if it beats downward it increases on downward gaze. A minimal vertical nystagmus however may be elicited only when the person looks far up or down. Vertical nystagmus is indicative of involvement of the vestibular nucleus or its immediate connections. When it beats upward the superior vestibular nucleus is implicated.

Rotary nystagmus does not change its direction with change of gaze. A clockwise nystagmus may increase slightly on gaze to the right and usually will indicate involvement of the vestibular nucleus on the right side. A counterclockwise nystagmus will increase in intensity slightly on gaze to the left and will usually be indicative of involvement of the vestibular nucleus on the left side.

If, on forward gaze, nystagmus is not present but if horizontal nystagmus is elicited when the patient looks to each side, this condition is caused by toxic effects from sedative drugs or by a disorder in the cerebellum. If weakness of gaze to either side is apparent, then the nystagmus may be paretic. When nystagmus appears only on extreme lateral gaze and when it is greater or is present only in the out-turning eye, such nystagmus must be regarded as physiologic and not of diagnostic significance.

If the movements of the two eyes are dissociated there is involvement of the medial longitudinal fasciculus or the neighboring reticulate substance. This may be manifested by internuclear ophthalmoplegia or monocular nystagmus, or in other instances paralysis of one of the extraocular muscles might be complicating the primary picture of nystagmus.

It is evident that several varieties of nystagmus are recognizable, each having special characteristics and arising through a disturbance in a particular location, which may be in the visual system, the labyrinth, the muscles of the eye, or the central nervous system. In spite of the failure of a few varieties to fit into this scheme and the occasional occurrence of complex combinations, a simple classification, such as the one presented, can aid in the interpretation of the various forms of nystagmus.

The Mayo Clinic.

References

1. Cogan, D. G.: Neurology of the Ocular Muscles. Springfield, Illinois, Charles C Thomas, 1948, chap. 9, pp. 127-167.

2. DeJong, R. N.: Nystagmus: An appraisal and a classification. Arch. Neurol. & Psychiat., 55:43-56 (Jan.) 1946.

3. Fox, J. C., Jr.: Nystagmus. Yale J. Biol. & Med., **1**:224-236 (Mar.) 1929.
4. Gorman, W. F., and Brock, S.: Nystagmus: Its mechanism and significance. Am. J. M. Sc., **220**: 225-233 (Aug.) 1950.
5. Kestenbaum, A.: Clinical Methods of Neuro-ophthalmologic Examination. London, William Heinemann Medical Books, Ltd., 1948, chap. 8, pp. 216-260.
6. Michaelson, I. C.: Nystagmus. Glasgow M. J. **127**:98-106 (June) 1937.
7. Spaeth, E. B.: Nystagmus: Its diagnostic significance. Arch. Ophth., **44**:549-560 (Oct.) 1950.
8. Spiegel, E. A., and Sommer, I.: Neurology of the Eye, Ear, Nose, and Throat. New York, Grune & Stratton, 1944, pp. 103-123.
9. Fox, J. C., Jr.: Disorders of optic nystagmus due to cerebral tumors. Arch. Neurol. & Psychiat., **28**:1007-1029 (Nov.) 1932.
10. Harman, N. B.: Discussion on nystagmus. Proc. Roy. Soc. Med. **7**(pt. 2):56-61 (Neurol., Ophth. & Otol. Sect.), (Feb.) 1914.
11. Bárány, R.: Ueber die vom Ohrlabyrinth ausgelöste Gegenrollung der Augen bei Normalhörenden, Ohrenkranken und Taubstummen. Arch. f. Ohrenh., **68**:1-30 (May) 1906.
12. Mygind, S. H.: How does the caloric nystagmus arise? J. Laryng. & Otol., **40**:444-450 (July) 1925.
13. Mygind, S. H., and Dederding, D.: Ménière's disease as an indicator of disturbances in the water metabolism, capillary function, and body condition. Ann. Otol., Rhin. & Laryng., **47**:55-62 (Mar.) 1938.
14. Northington, P.: The disease of the vestibular system. In: Fowler, E. P., Jr.: Medicine of the Ear. New York, Nelson, 1947, chap. 12, pp. 423-496.
15. Lindsay, J. R.: The significance of a positional nystagmus in otoneurological diagnosis. Laryngoscope, **55**:527-551 (Oct.) 1945.
16. Nylén, C. O.: Positional nystagmus: A review and future prospects. J. Laryng. & Otol., **64**: 295-318 (June) 1950.
17. Riley, H. A.: The central nervous system control of the ocular movements and the disturbances of this mechanism. Arch. Ophth., **4**:885-910 (Dec.) 1930.
18. Holmes, G.: The symptoms of acute cerebellar injuries due to gunshot injuries. Brain, **40**:461-535 (Dec.) 1917.
19. Spiegel, E. A., and Sommer, I.: Neurology of the Eye, Ear, Nose, and Throat. New York, Grune & Stratton, 1944, p. 115.
20. Cogan, D. G.: Neurology of the Ocular Muscles. Springfield, Illinois, Charles C Thomas, 1948, p. 158.
21. Bender, M. B., and Weinstein, E. A.: Effects of stimulation and lesion of median longitudinal fasciculus in monkey. Arch. Neurol. & Psychiat., **52**:106-113 (Aug.) 1944.
22. Harris, Wilfred: Ataxic nystagmus: A pathognomonic sign in disseminated sclerosis. Brit. J. Ophth., **28**:40-42 (Jan.) 1944.
23. Cogan, D. G., Kubik, C. S., and Smith, W. L.: Unilateral internuclear ophthalmoplegia: Report of eight clinical cases with one postmortem study. Arch. Ophth., **44**:783-796 (Dec.) 1950.
24. Arkin, W., and Sterling, W.: Le nystagmus monoculaire. Arch. d'ophtal., **43**:77-82 (Feb.) 1926.
25. Rucker, C. W.: Seesaw nystagmus associated with choroiditis and positive neutralization test for toxoplasma. Arch. Ophth., **35**:301-302 (Mar.) 1946.
26. Sorsby, A.: Genetics in Ophthalmology. London, Butterworth, 1951, 251 pp.
27. Lyle, T. K.: Latent nystagmus. Tr. Ophth. Soc. U. Kingdom, **68**:187-195, 1948.
28. Sorsby, A.: Latent nystagmus. Brit. J. Ophth., **15**:1-18 (Jan.) 1931.
29. Llewellyn, L.: The relation of miners' nystagmus to general nystagmus. Proc. Roy. Soc. Med., **7**(pt. 2):42-48 (Neurol., Ophth. & Otol. Sect.), (Feb.) 1914.
30. McCord, C. P.: Occupational nystagmus in train dispatchers. J.A.M.A., **96**:1131-1134 (Apr.) 1931.
31. Bender, M. B., and O'Brien, F. H.: The influence of barbiturates on various forms of nystagmus. Am. J. Ophth., **29**:1541-1552 (Dec.) 1946.

NOTES, CASES, INSTRUMENTS

THE BLASKOVICZ PTERYGIUM OPERATION*

J. William Rosenthal, M.D.
New Orleans, Louisiana

No previous description of the pterygium operation of Blaskovicz has been found in the English literature, and it is presented here more for historic interest than to recommend its use.

The procedure was taught to my father, Dr. Jonas W. Rosenthal, by Dr. Lazlo Blaskovicz, chief of the Second Eye Clinic of Budapest, Hungary, about 1922 to 1926.

Technique

With the patient prepared and draped, a speculum is inserted between the lids, separating them. Using a von Graefe fixation forceps, the neck of the pterygium is grasped, causing tension while the head and neck of the growth is completely cut from the cornea and limbus with a Wiener keratome.

A Stevens scissors is then used to cut the upper and lower borders of the pterygium from the adjoining conjunctiva, from the limbus to the base of the pterygium. The body of the growth is separated by blunt dissection from the subconjunctival tissue.

A mosquito hemostat is placed across the pterygium neck and it is held taut while the suture of 4-0 black silk or chronic catgut is placed (fig. 1). The neck of the pterygium is then cut from the body just proximal to the hemostat. By tightening and tying the suture, the pterygium is effectively rolled up near the medial canthus.

Blaskovicz left the denuded areas to granulate in, but I have modified the procedure by undermining the conjunctival edges above and below, and approximating them with two sutures (fig. 2). The speculum is then removed and an eyepatch is applied.

Case reports

Twenty-one patients have been operated using this technique. Their ages ranged from 24 to 76 years, with an average of 49 years. Seven were female and 14 male patients. Twenty pterygia were primary; one was recurrent. Postoperatively there were 14 (67 percent) cures, six (28 percent) recurrences, and one patient failed to return for observation. Follow-up periods ranged from two to 15 months.

Complications

In only two cases was there a complication and it was identical. A mass of granula-

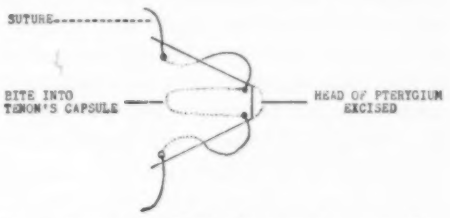


Fig. 1 (Rosenthal). Blaskovicz pterygium operation. Method of suture insertion.

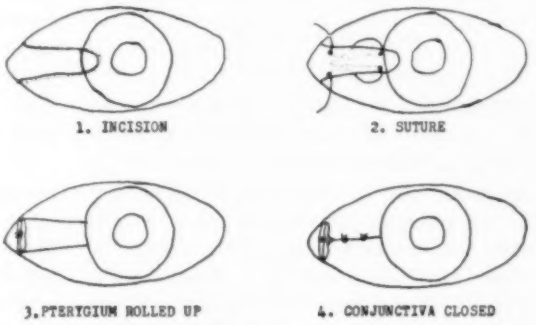


Fig. 2 (Rosenthal). Blaskovicz pterygium operation.

* From the Charity Hospital of Louisiana.

tion tissue appeared, adjacent to the limbus where the body of the pterygium had approximated the sclera.

Both of these cases were treated by removal of the mass, cauterizing the base with phenol, and overlapping the area with undermined conjunctiva.

The two cases were done early in the series—before I had modified the procedure by covering the denuded areas with conjunctiva.

Comment

One cannot adequately evaluate the Blaskovicz operation on such a small group of patients, but certain impressions were gained that should be recorded.

The eyes operated by this method remained red and irritable for several weeks longer than those operated by the McReynold's method. That is, the pterygium, rolled in the medial canthus, presented a red bulging mass that did not atrophy for many months.

In addition, there was rough granulation tissue covered by mucus and a pool of tears where the conjunctiva could not be completely closed over the episcleral tissue next to the rolled-up pterygium. Therefore, it seems that the Blaskovicz procedure gives less desirable cosmetic results than does the McReynold's operation.

ACUTE CONJUNCTIVITIS WITH LYMPH-GLAND SUPPURATION

John A. Egan, M.D.
Denver, Colorado

A scanty survey of the literature reveals that preauricular-gland suppuration complicating an acute bacterial conjunctivitis is somewhat rare. A canvass of the physicians of the Colorado Ophthalmological Society, about 40 members, revealed that none had even seen such a condition. Undoubtedly, many ophthalmolgists have not seen such an infection; therefore, the following case history seems worthy of record.

Case report

Mrs. J. C., aged 31 years, a white woman, reported to my office on January 31, 1952, with a very acute, severe, seropurulent conjunctivitis of the right eye. There was considerable edema of the lids, chemosis, and the discharge was profuse. The right preauricular lymph gland was palpable, about the size of a large pea. There was no staining of the cornea with fluorescein as seen with the biomicroscope. Otherwise, also, the eye was normal. The left eye appeared normal.

The patient gave a history of being on the street with a strong wind blowing when suddenly something blew into her right eye. When seen several hours later, the patient exhibited the clinical findings just described. I found no evidence of a foreign body.

Smears stained by Gram's method showed myriad gram-positive cocci in every field examined. Culture on blood agar resulted in a flourishing growth typical of hemolytic Staphylococcus aureus.

Treatment and further course

Aureomycin eye drops were prescribed once an hour together with cold compresses. The following day the conjunctivitis was improved, the preauricular gland was no larger. She continued to improve and the next day sodium sulfathiazole (eye ointment, five percent) was prescribed three times daily and the aureomycin was stopped.

On February 4, 1952, five days after the initial consultation, the patient returned to the office. The conjunctivitis was apparently cured but the right preauricular gland was large, swollen and tense. It was incised and about three to five cc. of yellow, lumpy pus were evacuated.

On February 12, 1952 the patient again reported, this time with apparently the very same condition starting in the left eye, although for the first few days of the original infection eye medication was used prophylactically in the left eye too. In addition to the virulent conjunctivitis this time, the left preauricular gland was swollen. It was about

the size of an almond and promised to suppurate. The right eye and gland were normal. Sodium-sulfathiazole ointment and cold compresses were again prescribed. In addition, however, aureomycin capsules (250 mg.) were given every four hours. This time the eye and gland conditions were cured, the latter without suppuration.

I regret that I did not take cultures of the pus from the right preauricular gland when I incised it, and also from the left eye, but I failed to realize at the time how rare the condition was.

1765 Sherman Street.

HOLE IN THE MACULA DUE TO SYPHILIS

Report of a case

W. A. Manschot, M.D.
Rotterdam, The Netherlands
AND
C. H. O. M. von Winning, M.D.
The Hague, The Netherlands

Formation of real holes in the macula is caused by: (1) A coalescence of cystoid spaces gradually leading to a complete breakdown of the tissue, and (2) retinal rupture due to shrinkage and traction of scar tissue in its vicinity (Zeeman[1]).

The latter cause of macular holes has been neglected in the more recent literature and Verhoeff,[2] for instance, states that a macular hole is always preceded by cystic degeneration.

The following case report shows a typical specimen of a macular hole caused by shrinkage and traction of nearby fibrous tissue. Moreover, the case is the first to be described in the literature, in which a macular hole is due to a syphilitic process.

Report of a case

A 44-year-old sailor (a Negro from Madagascar) complained that his right eye had become blind during his last journey.

Ophthalmic examination showed the left eye to be without any abnormality. Vision was 5/5 in the left eye and 1/300 in the right eye. The right eye showed no anomalies in the anterior segment but fundus examination disclosed a local periarteritis of the first large branch of the inferior temporal artery in the area just below the macula.

Fig. 1 (Manschot and von Winning). Reproduction of drawing showing hole in macula due to syphilis.

The fire part of the artery showed no abnormalities, except for a bright reflex, but very near its origin, the branch was covered by a white coat of fibrous tissue from which a branch pointed upward in the direction of the macula (fig. 1).

A little more peripherally the branch divided into two smaller branches and the periarteritic process ended just before this division. The whole length of the periarteritic process was no longer than two times the diameter of the disc. The peripheral part of the branch after the division showed no anomalies; the lumen had not been narrowed and the artery-wall was, except for its reflex, not visible.

In the macular region, a large, dark-red hole was visible, measuring almost one half of the diameter of the disc. The edges of the hole were clear, distinct, and within the hole some bright, white points of different sizes could be seen.

The retina showed radiating folds converging to the mass of fibrous tissue below the macula. Between the hole and the end of the branch of fibrous tissue that pointed to the macula, a semicircular dark line was

visible. Examination with the slitlamp revealed that this semicircular line was the expression of a local retinoschism.

General examination. The first heart sound was muffled and the second aortic sound was accentuated. The liver and the spleen were not palpable. Some inguinal lymph nodes were found. No scars of framboesia were visible.

The blood-pressure was 170/105 mm. Hg. The heart was not enlarged radioscopically but the shadow of the ascending aorta was too wide and too dark.

Laboratory studies revealed a 100-percent positive Wassermann test and a strongly positive Meinecke test. The rate of blood sedimentation was 28 mm. within the first hour and 42 mm. within two hours. The urea content of the blood was 450 mg./l.

Comment

There can be no doubt that syphilis was the cause of the retinal periarteritis in our case. This is proven by the highly positive blood tests and by the clinical aspect of the periarteritis. Both Igersheimer[3] and Haab[4] state that the syphilitic periarteritis is marked by its local character.

Even when the artery is enclosed by a thick layer of connective tissue, its lumen is not totally closed. The peripheral aspect of the artery always becomes more normal. Moreover, there can be no doubt that the hole in the macula is a consequence of the shrinkage and traction of the periarterial fibrous tissue. This traction also appears in the radiating folds in the retina, converging to the mass of fibrous tissue, as well as in the retinoschism between the edges of the macular hole and the end of the branch of fibrous tissue that points to the hole.

One question still must be solved, namely, the nature of the white points at the bottom of the macular hole. These white points are represented in many pictures of macular holes, for instance in the atlas of Wilmer, in the atlas of Haab, and in a recent article of Croll and Croll.[5] Wilmer states that these points resemble accumulations of cholesterin, but in the entire literature we could not find an anatomic study in which the nature of these points is properly described.

157 Nieuwe Binnenweg.
47 Josef Israëlstraat.

References

1. Zeeman, W. P. C.: Arch. f. Ophth., **79**:259, 1911.
2. Verhoeff and Grossman: Arch. Ophth., **18**:561, 1937.
3. Igersheimer, J.: Syphilis und Auge. Berlin, Springer, 1918.
4. Haab, O.: Festschrift für Helmholtz, 1891.
5. Croll, L. J., and Croll, M.: Am. J. Ophth., **33**:248, 1950.

MEMORY AID FOR THE PARALLAX TEST

Joseph I. Pascal, M.D.
New York

The cover-and-prism test for detecting and measuring heterophoria has its subjective counterpart in Duane's parallax test. As the occluder is passed from one eye to the other, while the patient fixes a target, the target seems to move one way or the other depending upon the kind of heterophoria present. It is a very delicate test and will disclose, in a good observer, an error of a fraction of a prism diopter. It is especially efficient for uncovering small amounts of hyperphoria.

There are several rules given for interpreting the kind of heterophoria from the direction of apparent movement of the target. These rules are easily forgotten. The simplest way to remember the relationship is to remember that *both* eyes always move like the target. Thus, for example, as the occluder (which may be a simple business card) is passed from the right eye to the left eye the target seems to move to the left, then *both* eyes have moved to the left during the process. If the target seems to move to the right, then both eyes have moved to the

right. If the target seems to move up then both eyes have moved up, and if the target seems to move down then both eyes have moved down.

Although both eyes move in the same direction, the nature of the movement is different in the two eyes. In the covered eye, the movement is a relaxing movement, allowing the eye to move to its position of rest. In the suddenly uncovered eye, the movement is a corrective movement, that is, a movement of redress in order to take up fixation.

Thus, suppose, as the card is moved from the left eye to the right eye, the target seems to move up, then both eyes have moved up, the (covered) right eye to its position of rest, the left eye to take up fixation. This shows a right hyperphoria, a tendency of the right eye to go up when freed from fusion impulses.

In this same case, when the card is now moved from the right eye to the left, the target will seem to go down. Both eyes have moved down, the right, exposed eye to take up fixation, the left, covered eye to its position of rest, showing a left hypophoria, which is a concomitant right hyperphoria.

Of course, exactly the same holds true in horizontal imbalances. If as the card is moved from the right eye to the left eye, the target seems to move to the left, then both eyes have moved to the left. The movement of the left eye behind the cover shows exophoria.

On moving the card in this case from the left eye to the right eye, the target will seem to move to the right as both eyes have moved to the right. The movement of the right eye behind the cover again shows exophoria.

A rule I often used for horizontal movements is that when the target seems to move *with* the card there is exophoria, when the target seems to move *against* the card there is esophoria. But the one single rule that both eyes always move the same as the target covers all cases and is the one most easily remembered and applied.

37 West 97th Street (25)

AN ELECTRODE FOR USE IN RETINAL SEPARATION*

WILLIAM J. HARRISON, M.D.
Philadelphia, Pennsylvania

In the operation to correct retinal separation, one may elect to use the electrode giving a single application at a time. This requires painstaking time.

Fig. 1 (Harrison). Electrode for use in retinal separation.

With this thought in mind, I have constructed an electrode shaped to conform to the eye and consisting of three insulated areas placed four mm. apart, so that one obtains three areas of surface coagulation with one application. It is best operated on the current furnished by the ophthalmotherm. The machine should be set from 350 to 500.

135 South 17th Street.

* This electrode may be obtained from the E. B. Meyrowitz Surgical Instruments Co., Inc., 520 Fifth Avenue, New York 18, New York.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

COLORADO OPHTHALMOLOGICAL SOCIETY

September 20, 1952

DR. FRITZ NELSON, *president*

REFRACTION PROBLEM

DR. FRITZ NELSON presented Mrs. J. H. B., aged 48 years, who was first seen on October 24, 1950, because vision had been failing for more than six months. An immature posterior cortical cataract was found in the right eye. A rather advanced cortical and nuclear cataract was found in the left eye.

Refraction was: R.E., −3.5D. sph. ⊂ −5.0D. cyl. ax. 90°; L.E., −3.5D. sph. ⊂ −5.0D. cyl. ax. 75°. Corrected vision was: R.E., 5/12, partly; L.E., 5/50. Removal of the cataract of the left eye was recommended.

Intracapsular extraction of the cataract on the left eye was performed on October 31st. The patient had an uncontrolled diabetic condition, but blood-sugar had been close to normal. At the time of the operation, it went up to 190 mg. percent in spite of a strict diet. However, by the middle of December, it had been reduced to normal.

On January 3, 1951, the patient was given a corrective lens for the left eye. With a +4.5D. sph. ⊂ +6.5D. cyl. ax. 172°, vision in the left eye was 5/5. The diabetic condition was kept pretty well under control.

On February 19, 1952, the patient was admitted to Glockner Hospital for extraction of the cataract from the right eye. Corrected vision in this eye had diminished to 5/50. This cataract was extracted in capsule and a small amount of fluid vitreous was lost. The postoperative course was uneventful. On April 16th, the right eye was refracted. With a +3.0D. sph. ⊂ +5.0D. sph. cyl. ax. 12°, vision was 5/3, partly. A few weeks later, the patient complained that she could see better in her right eye without the correction than with it.

Tests showed that she accepted much less astigmatic correction than objective refraction seemed to indicate. The reading on the Javal-Schiøtz ophthalmometer was:

	45.45
5	50.50

The astigmatic correction was reduced to +2.5D. cyl., but the patient still complained that she could see better without glasses than with.

On June 12th her uncorrected vision was found to be 5/5. With a +0.5D. cyl. ax. 10°, it was 5/3−. No further spherical or astigmatic correction was accepted by this eye in spite of the fact that the ophthalmometer reading still showed a corneal astigmatism of five diopters against the rule as present before the operation and, skiascopically, the same value was found.

The patient was again refracted on August 12th, and accepted only a +0.5D. cyl. ax. 10° combined with a +0.5D. sph. Skiascopically, however, the vertical value was still +9.0D. and the horizontal was +4.0D. The ophthalmometer reading showed the same amount of corneal astigmatism as before.

Discussion. Dr. Nelson said that he had had this patient's vision checked on three different charts and the objective refractive tests repeated. He was at a loss to explain the discrepancy between the subjective and objective findings.

CONGENITAL OCCLUSION OF TEAR DUCT

DR. FRITZ NELSON presented Marilyn B., a white girl, aged six years, whose parents gave the history that her eyes had been watering since birth but that they had been

advised not to take the child to an oculist before she reached the age of five years. Late in the spring of 1951, an oculist was consulted and he then irrigated the tear sacs and, late in the summer of that year, probed both ducts under general anesthesia.

The record did not show whether the pathways were irrigated after this procedure but the parents stated that both eyes and nostrils bled abundantly when the child was returned to the ward from surgery. Apparently, the ducts closed soon after the probing and neither epiphora nor pus was drained.

When first seen in April, 1952, tears and pus streamed from the child's eyes over her face. Irrigation revealed quantities of pus in both sacs and practically no drainage in either one. Both sacs were washed out repeatedly, first with chloromycetin and then with sulamyd (30 percent) until the infection cleared. No drainage was obtained, however.

The child was admitted to Glockner Hospital on May 21st. Under sodium pentothal anesthesia, both ducts were probed with Bowman probes, No. 1 and No. 2. An obstruction was found on the left side at the entrance of the canaliculus into the sac. After the canaliculus was dilated, the probe was inserted vertically. At the entrance of the sac into the tear canal, a strong, bony obstruction was found. It was pierced with considerable force. Another bony obstruction was found close to the lower orifice.

There was also a bony obstruction on the right side at the entrance into the canal. Another, very tough, bony obstruction was present at the lower orifice.

After probing on the right side, there was considerable hemorrhage through the nostril; on the left, blood appeared only in the conjunctival sac. The left duct was again probed and a third bony obstruction, about three mm. below the second, was broken. Irrigation into the nose was then possible. Both ducts were washed first with diluted hydrogen peroxide, then adrenalin, and then with saline solution until the bleeding was completely stopped.

That same evening, both ducts were again irrigated and good drainage was found. There was no trace of blood in the irrigation fluid. The child was sent home that evening. Both ducts were irrigated daily for three days. Drainage became normal and the irrigation fluid was clear. When the child was seen for the last time on July 17th, pathways and drainage were absolutely normal. There was no epiphora and no pus.

Discussion. In discussing this patient and the paper which he presented on "Management of congenital occlusion of the tear duct," Dr. Nelson urged early probing in such cases, preferably before the second month of life without anesthesia; after that period, under general anesthesia. He also emphasized the importance of subsequent thorough irrigation to remove every trace of blood in order to avoid another probing and to obtain patency.

Dr. Ralph W. Danielson advised more prolonged conservative treatment before the tear passages of infants were probed.

Daniel Franklin,
Secretary.

CHICAGO OPHTHALMOLOGICAL SOCIETY

March 17, 1952

DR. GLENWAY W. NETHERCUT, *president*

The Sucker Memorial Clinic was held at Cook County Hospital at 4:00 p.m.

SCIENTIFIC PROGRAM

MACULAR LESIONS OF VASCULAR ORIGIN

Part II: Functional vascular conditions leading to damage of the macula lutea. Dr. Bertha Klien presented this paper.

HEMANGIOMA OF THE ORBIT

DR. EDWARD C. ALBERS, in presenting

this paper, said that the subject of vascular tumors of the orbit never ceases to be interesting although the tumors occur infrequently. They may occur in any part of the body.

The best known classification of angiomatous tumors is that of Reese who divides them into four groups: (1) Capillary, (2) cavernous, (3) angioblastic, and (4) racemose or cirsoid.

Dr. Albers presented two cases. The first of a young man, aged 23 years, and the second that of an infant, aged three and one-half months. Slides were shown to illustrate these cases.

Discussion. Dr. Derrick Vail: Meningioma of the orbit is particularly intriguing. I am a believer in the intracranial approach to these lesions. Experience has bitterly convinced me that, with rare exceptions, the meningioma is not restricted to the orbit (slides).

With reference to Dr. Albers' presentation of hemangioma of the orbit, his removal of the tumor by surgery is logical. Not long ago Dr. Ralph Davis brought in a young woman in whom he had removed one of these hemangiomas at the age of five years. It became highly malignant and involved practically the whole side of the face with destruction of the eye.

A few months before that a woman, aged 32 years, was seen. She had a cavernous hemangioma with sudden onset of symptoms. At the time of examination it was obviously a multi-ocular growth. Some of it had seeded itself into the conjunctiva as little bits of blood tumor, and some of these had ruptured, giving bloody tears and chemosis on several occasions. The exophthalmos had progressed.

Even had X-ray therapy been suggested in this case, obviously the tumor was of such extent that not much would have been gained by its use. Diathermy was tried, using long needles properly insulated to destroy part of the tumor; however, this did not work very well.

Then the idea occurred that perhaps radon seeds would be advisable, if implanted simply through long needles with a trocar that pushed the seeds beyond the tip of the needle on all sides and below. In a few weeks there was obvious recession of the neoplasm and eventually the entire mass disappeared.

The eye resumed its proper place with no disturbance of vision or ocular damage. The patient has not been seen for two years, so whether there has been recurrence is not known.

While the essayist was fortunate in removing the first tumor, I do not feel that I would be entirely confident that all the tumor had been removed. Certainly with the second case I would be quite confident that the entire growth had been removed.

In these lesions one should remember that in the majority of instances they are much more extensive than one would expect. They may extend back into the orbit and spread like a cluster of grapes, and to remove the entire mass, and be sure of it, is a formidable project.

Dr. Edward C. Albers (closing): I agree with Dr. Vail that the method of treatment of these tumors is somewhat debatable. When a lesion like the one first described can be outlined, surgical removal is the method of choice. I do not feel confident that all the tumor has been removed in the first case, but it was felt that it could be treated by other means if it had not been entirely removed. Treatment by other means is difficult after surgery has been performed, but our Department of Dermatology felt that radium or radon therapy would be just as effective after surgery as before. Certainly the cosmetic results are much better following surgery than in most cases treated by other means.

Richard C. Gamble,
Secretary.

OPHTHALMOLOGICAL SOCIETY OF MADRID

June 6, 1952

Surgery of pterygium

Dr. Del Rio Cabanas read a paper on "The actual status of surgery of pterygium." After a brief historic survey, the speaker discussed the clinical and anatomicopathologic characteristics of pterygium, the pathogenesis, and the indications for operative intervention of the different varieties. He reviewed his own operative methods adopted after considerable experimental work on the use of conjunctiva from a cadaver. He said that the best methods were those which employed conjunctival grafts of whatever form, since these gave rise to the least number of recurrences.

Discussion. Dr. Marin Amat said that the pterygium is a special, paradoxal affection because, while it shows a simple, apparently unimportant, lesion, there are cases which present serious complications, such as pigmented nevus in a pterygium, or cystic degeneration in a ptergygium, as well as such neoplasms as epithelioma-sarcoma (white and pigmented).

Not only is the pterygium a paradoxal affection, it is also an enigmatic one. We do not know its true biologic essence. There are many which do not recur after extirpation; on the other hand there are many which do, which gives to the affection a malignant feature. There is also a marked incongruity between the pathologic anatomy and the vitality of a pterygium. The former gives it the character of a simple, hyperplasia of the conjunctival tissue, while the latter marks a strong tendency to reproduce itself, although it does not show any metastasis.

Possibly Redslob is right in considering pterygium a benign neoplasm, similar to the fibromas of the abdominal wall which show a marked tendency to spread, but produce no metastases.

In addition to the tendency of the pterygium to recur after removal, the little operation may be followed occasionally by serious hemorrhage.

In order to prevent, as much as possible, the tendency to recurrence, the following two measures have been found most effective. First, complete extirpation, especially of the apex, where is found what Fuchs called the progressive or invasive zone; second, perfect coaptation of the lips of the lost conjunctival substance.

Dr. Mario Esteban said: I find it better to avoid all cutting instruments in removing the pterygium from the cornea. I grasp the pterygium in its entire thickness with forceps at the level of the limbus, introducing a hook under it, flush with the limbus. With gentle traction movements I slide the handle of the instrument until the pterygium is completely detached from the cornea. This is a simple maneuver which leaves the surface perfectly smooth and regular. The excision of the pterygium to the base is then finished with scissors.

The loss of conjunctival substance is generally repaired by a simple sliding and suture of the conjunctiva, freed above and below. One must be careful not to carry the sutures so near to the cornea that the conjunctiva slides over it. Many pterygiums which are said to recur are really pseudopterygia produced by a proliferation and extension of the conjunctiva over the lost corneal susbtance.

I have never used a graft of buccal mucous membrane. I have been requested by some patients who had such grafts made to remove them by another operation, saying that they would have preferred keeping the pterygium, rather than the thick, reddish transplant. For this reason, it seems to me the idea of Dr. Rio Cabanas to use conjunctiva from a cadaver rather than buccal mucous membrane is very plausible. I congratulate him on the idea and hope to hear the results of his experience with this procedure which has great promise.

Dr. Valentin-Gamozo Fernandez: It seems to me that suturing the conjunctival lips when making a complete extirpation of the

pterygium by Dr. Marin Amat's technique of anchoring the sutures in the sclera does not prevent the conjunctiva from tearing. The only thing which fixing the stitch in the sclera accomplishes is to change the direction of the opposing forces which result from the traction of the silk at the knot. It does not reënforce the borders of the conjunctiva through which the sutures pass, thus not preventing the rupture of the same.

In his reply Dr. Del Rio Cabanas said that malignant and cystic degenerations of the pterygium are well known because of the magnificent anatomicopathologic description by Fuchs. I believe we must cease to think that, if we make a conjunctiva-conjunctiva suture, both flaps, as they more or less retract, are subject to two opposing forces at this point, one pulling upward and one pulling downward. If we make the suture with a scleral anchorage, the same forces working against a fixed point in the sclera will relieve each conjunctival flap from the retracting effect and will lessen the possibility of tearing at the suture points.

There are several techniques which do not use conjunctival sutures after excision of a pterygium. These methods and the reasons the authors give for their procedures are covered in my paper. I have used these methods and I believe that the techniques which use conjunctival sutures give better results than those which do not use sutures. However, I agree that the matter of suturing or not suturing is of little importance with reference to the possibility of a recurrence. We must not forget that cicatrization leaves an indelible anatomic mark and this may be important with reference to a future recurrence.

Certainly most pterygiums operated on are small and do not show a tendency to recur. In these, any technique gives excellent results. It is the very large pterygiums which present surgical problems, although they almost always can be treated with a conjunctival tag either by traction or by a pedicle, because of the great elasticity of the conjunctiva. However, there are cases in which we do not have enough conjunctiva to cover the loss of substance. Then we must look for tissue from some other zones.

Frankly, I have seen the results obtained by the use of mucous membranes (generally labial) and they did not impress me favorably. For this reason I began using conjunctival tissue from the cadaver, and so far I am satisfied with the results, both from the standpoint of appearance and from the standpoint of a recurrence. The pterygiums operated on and treated by the old methods gave a recurrence in one case of three, but until now none of those operated on by the method I propose has recurred, although I may be premature in my claims as there has not yet been a sufficiently long time of observation.

I do not believe, as Dr. Munoz Poto claims, that recurrence of a pterygium is due to the fact that the patient returns to the occupation and environment (heat, dust, and so forth) in which he developed the pterygium in the first place. I rather think that recurrence is a natural propensity of pterygiums. French authors, who praise lamellar grafts in the treatment of pterygiums use cortisone and other methods in order to prevent vascularization at the base of the graft, and they have observed that the cases thus treated, fundamentally with beta-ray therapy, recur less often than do those not treated.

Joseph I. Pascal,
Translator.

AMERICAN JOURNAL OF OPHTHALMOLOGY

Published Monthly by the Ophthalmic Publishing Company

EDITORIAL STAFF

DERRICK VAIL, *Editor-in-Chief*
700 North Michigan Avenue, Chicago 11

LAWRENCE T. POST, *Consulting Editor*
640 South Kingshighway, Saint Louis 10

WILLIAM L. BENEDICT
100 First Avenue Building, Rochester, Minnesota

FREDERICK C. CORDES
384 Post Street, San Francisco 8

SIR STEWART DUKE-ELDER
63 Harley Street, London, W.1

EDWIN B. DUNPHY
243 Charles Street, Boston 14

F. HERBERT HAESSLER
561 North 15th Street, Milwaukee 3

PARKER HEATH
243 Charles Street, Boston 14

S. RODMAN IRVINE
9730 Wilshire Boulevard,
Beverly Hills, California

JAMES E. LEBENSOHN
4010 West Madison Street, Chicago 24

DONALD J. LYLE
601 Union Trust Building, Cincinnati 2

WILLIAM A. MANN
30 North Michigan Avenue, Chicago 2

P. ROBB McDONALD
1930 Chestnut Street, Philadelphia 3

JOHN V. V. NICHOLLS
1414 Drummond Street, Montreal

ALGERNON B. REESE
73 East 71st Street, New York 21

PHILLIPS THYGESON
220 Meridian Road
San Jose 26, California

M. URIBE TRONCOSO
500 West End Avenue, New York 24

ALAN C. WOODS
Johns Hopkins Hospital, Baltimore 5

KATHERINE FERGUSON CHALKLEY, *Manuscript Editor*
Lake Geneva, Wisconsin

Directors: WILLIAM L. BENEDICT, President; FREDERICK C. CORDES, Vice-President; WILLIAM A. MANN, Secretary and Treasurer; F. HERBERT HAESSLER, LAWRENCE T. POST, DERRICK VAIL.

Address original papers, other scientific communications including correspondence, also books for review to *Dr. Derrick Vail, 700 North Michigan Avenue, Chicago 11, Illinois;* Society Proceedings to *Mrs. Katherine F. Chalkley, Lake Geneva, Wisconsin.* Manuscripts should be *original copies,* typed in *double space,* with wide margins.

Exchange copies of medical journals should be sent to *Dr. F. Herbert Haessler, 561 North 15th Street, Milwaukee 3, Wisconsin.*

Subscriptions, application for single copies, notices of changes of address, and communications with reference to advertising should be addressed to the *Manager of Subscriptions and Advertising, 664 North Michigan Avenue, Chicago 11, Illinois.* Copy of advertisements must be sent to the manager by the 15th of the month preceding its appearance.

Change of address notice should be received not later than the 15th of the month prior to the issue for which the change is to go into effect. Both old and new addresses should be given.

Author's proofs should be corrected and returned within forty-eight hours to the *Manuscript Editor, Mrs. Katherine F. Chalkley, Lake Geneva, Wisconsin.* Twenty-five reprints of each article will be supplied to the author without charge. Additional reprints may be obtained from the printer, the George Banta Publishing Company, 450-458 Ahnaip Street, Menasha, Wisconsin, if ordered at the time proofs are returned. But reprints to contain colored plates must be ordered when the article is accepted.

HERPES CORNEAE

In 1936, Gundersen referred to herpes corneae as the most important specific keratitis in the United States. If this was true in 1936, it is more than ever true in 1953. Prior to the onset of World War II, I had observed only one case of bilateral dendritic keratitis in 14 years of ophthalmologic experience, but during and since the War, I have seen numerous bilateral cases and recently saw three in a single week.

It is my impression, moreover, that the severity of the dendritic lesions has increased steadily and that there is a higher incidence of such associated lesions as disciform keratitis and uveitis. There seems also to have been an extraordinary increase in the number of cases of primary ocular herpes in which the severe conjunctivitis and preauricular adenopathy have resembled the changes seen in epidemic keratoconjunctivitis.

Most significant of all, perhaps, is the fact

that, prior to the advent of cortisone, corneal perforation and hypopyon ulcer were unheard-of complications in herpes corneae; I have now seen two cortisone-treated cases with hypopyon and have heard of three with corneal perforation.

During the past 25 years, there has accumulated a great deal of knowledge on the epidemiology, the immunology, and the biologic and morphologic properties of herpes simplex virus, while little or no progress has been made in the field of therapy. Early hope that aureomycin, or other broad-spectrum antibiotics, would be effective has faded, and attempts at hyperimmunization for the prevention of recurrences have in general been unsuccessful. The only hope for therapy still rests in the local destruction of the virus in the affected tissues.

What are the prospects for therapeutic advance in herpes? Since the typical viruses are intracellular microörganisms, dependent on host cells for their principal metabolic functions, the best hope lies in finding a way to interrupt by chemotherapy some essential process. This technique has, of course, been applied with outstanding success to bacteria, which are free-living, to the Rickettsiae, which are intracellular, and to the atypical viruses of the psittacosis-trachoma-lymphogranuloma venereum group, which are also intracellular.

At the time of writing there is, as yet, no clear-cut example of successful chemotherapy in a typical virus disease, but the future should not be considered too black. Certainly the importance of virus diseases in general is such that an intensive search for effective chemotherapeutic agents will continue to be made; so far as ophthalmology is concerned no more important problem exists than the virus disease under discussion.

While awaiting therapeutic advances, efforts should be made to interrupt the various trigger mechanisms which are in many cases concerned with recurrences. The use of antipyretics and antihistaminics is certainly worthy of trial when fever is a precipitating cause or when such other activating mechanisms as sunburn, menstruation, emotional crisis, and allergy are known to be concerned.

The evidence is now accumulating from both the clinics and the laboratories that cortisone has a masking effect only in this disease. In spite of the fact that herpetic patients often feel more comfortable while taking cortisone, and that a few isolated reports of favorable effects have appeared, only harm can be expected from its widespread use. There are now on record a number of instances in which dendritic keratitis has developed in patients undergoing systemic or topical cortisone therapy, and it should be emphasized that all five of the very severe cases referred to in paragraph three had been cortisone-treated.

Phillips Thygeson.

INTERNATIONAL COUNCIL OF OPHTHALMOLOGY

The following is a short resume of the main business conducted by the International Council of Ophthalmology at its meeting held in Oxford on July 3 and 4, 1952.

There were present Duke-Elder (president—in the chair), Berens (vice-president), Hartmann (secretary), Alvaro (Brazil), Arganaraz (Argentine), Bietti (Italy), Law (Great Britain), Löhlein, (Germany), Samuels (United States), Velter (France), Bailliart (Committee for the Prevention of Blindness), MacCallan (League against Trachoma).

There were in attendance Copper (secretary for the Indicia), Fritz (secretary for internationalizations), Dunnington (vice-president of the International Congress 1954).

The deaths of van der Hoeve and Kapuściński were regretfully recorded and the resignations of Tooke and Nakamura received. In their places were elected Charamis (Greece), Duggan (India), and Stuart Ramsay (Canada).

In the internationalizations of visual standards the questions of the standardiza-

tion of optotypes, color vision, light sense, visual requirements for transport, and the standardization of tonometry were referred to their respective committees for further study. The study of the requirements for education in ophthalmology in different countries of the world was similarly referred.

The future of the Indicia was discussed and it was decided that a more comprehensive volume should be published at each International Congress, containing, in addition to a directory of ophthalmologists, a list of ophthalmologic hospitals and journals throughout the world, as well as extracts of the minutes and activities of the International Council. It was suggested that the volume should be sold at each International Congress and that its name should be changed in the interests of linguistic purity to Index Ophthalmologicus.

New statutes for the International Federation, the International Council, and International Congresses were further studied; these will be presented to the Federation at the International Congress in New York in 1954.

The University of Lausanne is again to present a Gonin Medal at this congress and it was agreed that the recipient chosen by the council should first give a lecture at the University of Lausanne before he receives the award at the International Congress.

The treasurer's report showed that a number of countries were in considerable arrears in their subscriptions to the International Council. It was confirmed that such countries should not participate in the deliberations of the federation unless their subscriptions were fully paid before 1954.

The International Dictionary of Ophthalmological Terms being prepared under the authority of the council will be published in Switzerland by Karger without financial responsibility on the part of the council.

The following decisions were made with regard to the International Congress of 1954. This congress will be held in the Waldorf-Astoria Hotel, New York City, from September 13 to 17, 1954, inclusive. The three administrative languages will be English, French, and Spanish. The two main subjects for discussion will be: (1) Primary Glaucoma—opened by Vail of Chicago (general considerations and etiology), Thiel of Frankfort (medical treatment), Sourdille of Nantes (surgical treatment); (2) Etiology of Uveitis—opened by Woods of Baltimore (general considerations), Ashton of London (allergic factors), and Cavara of Rome (the role of viruses).

The subscriptions for the congress will be: for those living outside the North American continent, full members 15 United States dollars; associate and scientific associate members, five United States dollars. The subscription for those living within the North American continent will be fixed by the American committee organizing the congress.

The next meeting of the council was fixed for Paris on June 20, 1953.

(NOTE. It may not be universally known that the International Federation is made up of a delegate from each national society and meets at each International Congress; the International Council is appointed by the International Federation and acts as its executive body.)

E. Hartmann,
Secretary.

XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

September 13 through September 17, 1954
New York City
Waldorf-Astoria Hotel
Subjects for discussion: *Glaucoma* and *Uveitis*

CORRESPONDENCE

RETROLENTAL FIBROPLASIA IN NEGROES

Editor,
American Journal of Ophthalmology:

In the excellent review article by Dr. Leona Zacharias (AMERICAN JOURNAL OF

OPHTHALMOLOGY, 35:1426 [Oct.] 1952), it was stated that in Cook County Hospital (one of the largest in the United States) there had been almost no retrolental fibroplasia. Dr. Zacharias concluded that, in this country at least, retrolental fibroplasia may be more common among whites than among Negroes.

Although, as this article pointed out, before June, 1951, there were almost no cases of retrolental fibroplasia discovered at Cook County Hospital, since then 30 cases of retrolental fibroplasia have been discovered. All but two were in Negro infants. Here is another statistical oddity to add to the already complex and confusing knowledge concerning retrolental fibroplasia.

(Signed) Edward E. Hart,
Chicago, Illinois.

BOOK REVIEWS

BIOMICROSCOPIE ET HISTOPATHOLOGIE DE L'OEIL. By Archimede Busacca. Zurich, Schweitzer Druck and Verlagshaus, 1952. Volume I, 480 pages, 221 black and white illustrations and 128 in color on 28 plates, table of contents, and index. Price: Not listed (quoted at $35.00 by Grune and Stratton, the American distributors).

For many years the injunction of the senior ophthalmologist to his students when discussing biomicroscopy was "Read Vogt, the rest is commentary." The novice was quickly impressed by the comprehensiveness of this text but was very often equally confused by the enormous number of uncorrelated clinical conditions described. This was in part remedied by the appearance in 1942 of Berliner's classic text in which he devoted a preliminary paragraph in most cases to the underlying pathologic process. Dr. Busacca has continued this trend and now offers us a text on biomicroscopy that is organized entirely along histopathologic rather than clinical lines. Indeed it is as much a text of histopathology as it is a treatise on biomicroscopy.

This first volume is divided into three parts: (1) General considerations, (2) Conjunctiva, (3) Cornea.

Under "General considerations," Busacca discusses at great length what we consider general pathology, that is, degeneration (which he terms "regressive metamorphoses"), inflammation, and so forth, and concludes this portion with an abbreviated discussion of the technique of biomicroscopy, abbreviated because, as Busacca states, "The technique of biomicroscopy is actually known by almost all oculists and it can be found explained in great detail in the classic modern texts."

Part two, "Conjunctiva," is subdivided into two sections: the first giving the anatomy and general pathology of the conjunctiva and the second discussing the special conjunctivitides and degenerations.

Part three is similarly subdivided into a first section containing the anatomy and general pathology of the cornea and the second section discussing the special keratitides (epithelial and interstitial), the dystrophies and degenerations, and concluding with a chapter on cicatricial lesions of the cornea.

Each of the pathologic groups is illustrated by many black and white microscopic sections, as well as by a considerable number of colored illustrations of the biomicroscopic appearance of the lesions being discussed. The organization of the material is excellent and, although some of the rare lesions, such as tumors of the conjunctiva, have been omitted, the more common pathologic entities are discussed in great detail. The colored plates are among the most beautiful seen by this reviewer, especially in regard to the brightness, freshness, and delicacy of hue obtained.

This is a large book, page size about 7½ by 10½ inches, and is printed on a heavy gloss paper which should retain its color values indefinitely. To the ophthalmologist who knows French this book will be a delight and to the stumbler in this language it will provide an excellent opportunity to brush up for that trip aboard that seems to be *de*

rigueur for ophthalmologists today. This book is a noteworthy addition to the small group of excellent texts in ocular pathology that have been appearing of late. This is all to the good since, to paraphrase Homer Smith, in this molecular world, structure always underlies dysfunction.

David Shoch.

TRANSACTIONS OF THE Société d'Ophtalmologie de France 1952, January, pp. 1-46.

H. A. Miller, J. Duchêne, and Carton used terramycin drops with success in the treatment of penicillin- and aureomycin-resistant staphylococcic conjunctivitis, corneal ulcers, dendritic keratitis, and herpetic keratitis. The etiologic diagnosis of herpetic keratitis was verified by rabbit inoculation. Subconjunctival terramycin injections are not recommended, in spite of their effectiveness, because they are painful and irritating. They proved to be helpful in a case of experimental Friedländer-bacillus endophthalmitis.

P. Desvignes and G. Sadoughi emphasized the difficulties in recognizing the etiology of proliferating chorioretinitis and presented several case histories to prove that tuberculosis is not always the cause.

Graciansky, Voisin, Grupper, and Landriev reported favorably on local cortisone applications, retrobulbar injection of novocain and 15 percent alcohol, and high doses of penicillin in the treatment of interstitial keratitis. In refractory cases they suggested lamellar keratoplasty.

Offret, Mme. Godde, and Mangue reviewed the pathogenesis and clinical picture of disciform keratitis. They considered aureomycin in large doses and subconjunctival cortisone injections to be the treatment of choice but the cortisone must not be given before the third week because of the possibility of a reactivation in a viral lesion.

DeJean and Bourdet presented a very interesting study and described a recurrence of Mooren's ulcer in a nearly total transplant of a perforating keratoplasty.

R. Weekers discussed the action of eserine and DFP and presented the different clinical phases of their action, in the tabular form.

Michael Onfray appraised color retinography not only for the interpretation of details and localization of the fundus lesions themselves but also in their relationship to perimetric findings, capillary studies, and general medical problems. He believes that routine, serial color retinography has great practical value.

Alice R. Deutsch.

ARCHIVOS DE LA ASOCIACIÓN PARA EVITAR LA CEGUERA EN MEXICO. Volume 7, 1949.

The seventh volume of the Archives, published in 1949 but distributed more recently, is a record of much interesting and well-documented work. A detailed statistical analysis of the refraction of school children in the city of Mexico is presented by L. Sanchez Bulnes, P. Cornejo Lastra, R. Figueroa Ortiz, R. Coutreras Ramírez, and G. Román y Carillo. The data are presented in tables and graphs. V. Ramírez Esteva contributes a study of new contact lenses. I. S. Tassman discusses the correction of astigmatism in asthenopia resulting from errors of refraction. P. C. Craig recommends trifocal lenses to be worn by the ophthalmic surgeon. They have a rectangular upper segment with optic centers nearer together than those of the lower segments.

Manuel de Rioas Cherif recognizes that, of the many ideas with which Professor Marguez has enriched the science of ophthalmology, one of the most brilliant is his method of coincidences. de Rioas Cherif presents a closely reasoned analysis of the application of this method in the diagnosis of spasm of the extraocular muscles. His exposition provides explanation in the utmost detail which is clarified with the help of excellent diagrams. Joseph Pascal demonstrates his simplified schemes for registration of the

ocular muscles and diplopia, which is based on the benzene ring.

E. G. López Quinones discusses the entire problem of trachoma—history, incidence, etiology, chemical forms, pathology, diagnosis, and treatment. H. Escapini describes the technique of keratoplasty and exemplifies his results by presenting the data and photographs in 10 cases.

D. Silva and V. Morales find pre-ophthalmometry invaluable in the diagnosis of keratoconus and, in another contribution, report 100 cases of keratoconus in which contact lenses were prescribed. Also reported are studies, with A. Guevara Rojas, of the endocrine functions in patients with keratoconus.

F. Palomino Dena, R. Soto, and L. Villegas León discuss the problem of toxoplasmosis and illustrate the clinical data with roentgenograms and fundus pictures, one of which is in color. E. Lopez Quiñones presents a case of central serous chorioretinitis illustrated with two plates in color and discusses the nature and treatment of the disease. He also describes an eye enucleated because of pseudoglaucoma and comments on the diagnostic problems.

L. Vergara Espino discusses the development of choroidal rupture. D. Silva and V. Morales describe their experience with a patient with Harada's disease, document their findings with reproductions of fundus photographs, colored plates of drawings of the fundus, and fields, and discuss the nature of the disease.

L. Sanchez Bulnes and D. Silva present a case of Vogt-Koyanagi syndrome in which bilateral symmetric staphyloma developed. Histologic preparations demonstrated that the disease was not the result of tuberculosis, syphilis, or sympathetic ophthalmia.

D. Silva offers an extensive discussion of the problems of retinoblastoma—the clinical manifestations, diagnosis, prognosis, and therapy. M. Puig Solanes offers a classification of hypertensive retinopathies with drawings of the fundus and tabulations of data.

The lesions of the optic nerve that arise in the course of the treatment of syphilis by means of trivalent arsenicals is discussed by L. Vergara Espino, who also outlines the ophthalmologic and neurologic diagnostic procedure which is necessary for proper management.

There is only one discussion of glaucoma in this volume. C. Espildors Lugue and J. Vizarra Ch. describe their technique of nonperforating cyclodiathermy and report the results of 71 operations in several types of glaucoma.

The diagnosis and treatment of intraocular foreign body is discussed in general and illustrated with 29 reports of cases by R. A. Chaviro. D. Kravitz discusses the diagnosis and treatment of mucocele of the frontal sinuses and reports four cases. F. Martinez Hinojosa describes a modification of Barraquer's operation, an exenteration of the eyeball with implantation of fat. D. Silva and M. Barojas report a case of osteoma of the orbit. Nine figures display the appearance of the patient, the fundus, roentgenograms, the excised neoplasm, and photomicrographs.

The cause and prevention of fractures in dacryocystorhinostomy are discussed by M. P. Palmer. R. Olivera Lopez reports his experience with dacryocystorhinostomy in children and adolescents and describes his techniques in detail. The description is made clear with help of 11 excellent diagrammatic drawings. The operation achieves the same results as in the adult.

F. H. Haessler.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Gsell, O. **Leptospiroses and the eye.** Klin. Monatsbl. f. Augenh. **120**:449-469, 1952.

Leptospiroses are becoming more and more important for the ophthalmologist. The most important late complication is uveitis; conjunctivitis occurs during the acute stage and optic neuritis may occur during the second febrile stage. All leptospiroses consist of an initial septicemia followed by a second febrile stage with involvement of a specific organ. Hyperemia of the conjunctiva in the first stage is frequent and of diagnostic importance. Optic neuritis or papilledema occurs during the second stage if there is meningitis. The iridocyclitis, which is a late complication, may be 1. acute and benign, 2. chronic and severe with a dense vitreous membrane, or 3. recurrent.

The frequency of uveitis in leptospirosis varies from 2 to 20 percent. It has reached 40 percent for Weil's disease in Japan. The pathogenesis is not well understood, but we can assume that leptospirillae reach the eye via the bloodstream. More important for the diagnosis are antibodies in the serum and the aqueous. Ophthalmia lenta and Behçet's syndrome have been regarded as a leptospirosis (Gilbert), but the evidence is inconclusive. It is interesting to note that the periodic panophthalmitis of horses is definitely a leptospirosis. The prognosis of the iridocyclitis is usually good. Antibiotics are effective. (4 figures, 105 references)

Frederick C. Blodi.

Lepri, Giuseppe. **Observations with the phase contrast microscope on the cellular elements of the conjunctival and corneal epithelium in epidemic keratoconjunctivitis.** Gior. ital. oftal. **5**:314-323, July-Aug., 1952.

While the normal cell structure could be seen clearly, no intracellular or intranuclear inclusion bodies were visible in unfixed, unstained preparations. These negative findings do not prove their absence but further studies of this and other diseases associated with inclusion bodies should be made. (6 figures, 16 references)

John J. Stern.

Nakashima, Nobuyuki. **Experimental opticochiasmatic arachnoiditis.** Acta Soc.

Ophth. Japan **56**:1019-1027, Sept., & 1187-1194, Oct., 1952.

The toxin of bacillus coli was introduced into the carotid artery of rabbits which had been sensitized with B.C.G. and a severe opticochiasmatic arachnoiditis was brought about. There were severe subarachnoidal hemorrhage, new growth of blood vessels, perivascular inflammations about arterioles and capillary vessels and venous congestion; the arachnoid looked like a hematoma. These findings were particularly pronounced 30 to 90 days after the injection of the toxin. The author considers these changes to be a parallergic reaction, and the experiment to give some indication of the etiology of acute retrobulbar optic neuritis.

Yukihiko Mitsui.

Oesterle, Gabriella. **Pathogenic, aerobic, sporeforming microorganisms in ophthalmology.** Klin. Monatsbl. f. Augenh. **120**:246-254, 1952.

The author observed two cases of postoperative endophthalmitis caused by Bacillus sphaericus, an aerobic sporeformer. She discusses the role of aerobic sporeforming microorganisms in ocular diseases. In nearly 80 percent of the normal rabbit eyes which she examined and in 3.5 percent of normal human conjunctivas aerobic sporeformers could be found. The pathogenicity of these microorganisms could be shown by intracorneal injections into rabbit eyes. The author believes that these microorganisms belong to the normal flora of the conjunctiva. They are potentially dangerous when carried into the interior of the eye. There is no effective drug against them and the best prophylaxis is a careful mechanical cleansing of the conjunctival sac before the operation. (3 tables, 17 references)

Frederick C. Blodi.

Ogino, S., Yamada, Y., and Ichihara, F. **Biochemical study of cataract.** Acta Soc. Ophth. Japan **56**:1170-1174, Oct., 1952.

When adrenalin (0.3 cm. of 0.05 percent solution) was introduced into developing chick embryos, a cataract rarely resulted, while an introduction of adrenochrome was followed by a development of cataract in 12 of 18 embryos when introduced on the fifth or sixth day of incubation. The authors consider a high redox potency of adrenochrome to be responsible for the cataract development. Ascorbic acid (2 mg.) and anthranilic acid (2 mg.) prevented the development of cataract when introduced into the embryo before the use of adrenochrome, while glutathione had no preventive effect. In the latter half of the incubation period (from the twelfth to the sixteenth day), adrenochrome rarely caused cataract. The authors ascribe this to the production of ascorbic acid in the embryo in the latter half of the incubation period.

Yukihiko Mitsui.

Schlossberger, H. **Viruses, rickettsias, toxoplasma, leptospiras.** Klin. Monatsbl. f. Augenh. **120**:225-236, 1952.

This is a short survey on some of the newer aspects of these microorganisms given by an epidemiologist for ophthalmologists. The author discusses especially Q-fever, rickettsial pox and the tests for toxoplasmosis. The leptospiroses are discussed in greater detail. It could be shown (Langbein) that ticks can transmit leptospira, as in Weil's disease.

Frederic C. Blodi.

Yusa, Mitsuru. **Influence of some substances administered intracisternally to the optic nerve.** Acta Soc. Ophth. Japan **56**:1014-1019, Sept., 1952.

The author studied the histopathologic changes in the optic nerves of rabbits after intracisternal administration of such substances as salvarsan, mercuric chloride, mercuric oxycyanide and five antibiotics. Salvarsan caused little change in the optic nerve; mercuric chloride and mercuric oxycyanide caused noninflam-

matory degenerative changes in the medullary sheath. Penicillin caused slight degenerative changes in the medullary sheath. Chloramphenicol caused a slight increase in the cellular elements of the arachnoid. Aureomycin caused a slight increase in cells of the arachnoid and degenerative changes and granulation tissue changes in the medullary sheath. Streptomycin showed no harmful effect.

Yukihiko Mitsui.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Auricchio, G., and Sbordone, G. **Glutaminic acid and retina.** Gior. ital. oftal. **5**:387-394, Sept.-Oct., 1952.

Six rabbits received daily intravenous injections of 4-percent sodium iodate. Six others of equal weight and color received the same treatment and, in addition, they received a daily dose of 100 mg. of the sodium salt of glutaminic acid intravenously and the same doses intramuscularly during the three days preceding, and during the administration of the sodium iodate. In the first group, the first signs of choroidal disturbance apeared after three to four days, in the second, after six to seven days. Two to three days after the first appearance of these signs, both groups exhibited typical pigmentations, similar to human retinitis pigmentosa. Histologic studies showed that the retinas of animals treated with glutaminic acid were subjected to less structural changes than those of animals receiving the iodine salt alone. Treatment of ainmals in which fundus changes had been previously produced by sodium iodate, with the same doses of glutaminic acid, failed to change the fundus or the histological appearance. The experiments prove a protective action of glutaminic acid against the toxic effect of sodium iodate on the retina. (2 figures, 57 references) John J. Stern.

Bocci, G. **Primary sensitization of the conjunctiva against tuberculous antigen.** Boll. d'ocul. **31**:459-462, Aug., 1952.

In continuation of former investigations, Bocci injected killed tubercle bacilli or the diagnostic preparation anatuberculin intravenously 15 days after sensitization of the conjunctiva, at the time when the intradermal tuberculin reaction of the rabbit was still negative. Development of a purely local allergy in the rabbit's conjunctiva was observed. (9 references) K. W. Ascher.

Bonavolonta, G., and De Berardinis, E. **The action of cortisone upon processes of cicatrization of corneal lesions and upon experimental corneal vascularization.** Rassegna ital. d'ottal. **21**:225-241, July-Aug., 1952.

The authors studied the effect of local application of cortisone in the process of healing experimentally-induced wounds in the cornea of rabbits. The wounds were superficial, deep, and through the whole corneal thickness. Examination by vital staining with fluorescein, by the biomicroscope, and by histologic studies show that the local application of cortisone had a retarding effect, especially in deep wounds.

In a second group of rabbits, corneal vascularization was produced by the daily application of absolute alcohol, as proposed by Ehler. Here cortisone led to a more rapid and complete regression of the new-formed vessels. The results of treatment in laboratory animals corresponded exactly to those seen in clinical practice. (9 figures, 22 references)

Eugene M. Blake.

Brand, I. **Clinical experiences with a new synthetic mydriatic.** Klin. Monatsbl. f. Augenh. **120**:596-605, 1952.

This is a clinical evaluation of "Labotropin," a parasympathicolytic drug which in 1-percent solution easily brings about full dilation of the pupil without paralysis of accommodation. The intraocular pres-

sure did not show any changes. (4 figures, 10 references) Frederick C. Blodi.

Cascio, Giuseppe. **Demonstration of the anti-hyaluronidase activity of Tocopherol (Vitamin E) with irido-ciliary permeability test. (Experimental investigations.)** Gior. ital. oftal. **5**:423-434, Sept.-Oct., 1952.

After he had established that hyaluronidase increases the permeability of the blood-aqueous barrier, the author injected rabbits with 20 mg. of vitamin E subconjunctivally or 200 mg. intravenously for a period of 10 days. The increase of permeability produced by hyaluronidase was shown to have been reduced. (3 figures, 3 tables, 8 references) John J. Stern.

Gemolotto, Guiseppe. **Study of the glucose and ascorbic acid content in chorioretinal degeneration experimentally produced by iodate of sodium.** Rassegna ital. d'ottal. **21**:263-271, July-Aug., 1952.

In rabbits with chorioretinal degeneration induced by the use of sodium iodate, the writer determined an elevation of the percentage of glucose and a lowering of the value of ascorbic acid with a relative increase of the basic pH, but no other change in the blood. To explain the probable hypothesis biochemically, it is assumed that an increase of glucose which depends upon altered retinal function could affect the possible reduction of ascorbic acid into glucose. It is assumed that the sodium iodate develops an elective action upon ocular tissue. (4 tables, 21 references) Eugene M. Blake.

Grignolo, A., and Pannarale, M. **The influence of cortisone upon ocular tuberculosis.** Rassegna ital. d'ottal. **21**:242-253, July-Aug., 1952.

Research was conducted on the action of cortisone in experimental tuberculosis of the eye. The hormone was administered either locally, as an ointment, or parenterally to two groups of guinea pigs. The first group was composed of animals, originally free of tuberculosis, which received an inoculation of tubercle bacilli into the anterior chamber, while the second group was one of allergic-immune animals in which intraocular re-inoculation was practiced. The administration of cortisone, either locally or parenterally produced an aggravation and acceleration of the tuberculous process in animals of the first group, particularly in those treated by local application. No evidence of influence of any kind was observed in the tuberculous process of the immune-allergic guinea pigs. (2 figures, 26 references) Eugene M. Blake.

Higuchi, M. **Vitamin C synthetic action of the crystalline lens. II.** Acta Soc. Ophth. Japan **56**:1217-1221, Nov., 1952.

The synthetic action of beef lens in the production of vitamin C was studied as in the work previously reported (Am. J. Ophth. 35:1521), using 5-keto-d-gulonic acid as a starting material, with positive results. The synthetic ferment was inactivated either by heating or dialysis. However, the heat-inactivated ferment was made active when the ferment inactivated by a dialysis was added. The author concludes, therefore, that the ferments consist of two fractions: 1. a dialytic-thermostable and 2. a nondialytic-thermolabile fraction. The synthesis was accelerated by KCN, but impeded by $AgNO_3$. The experiments further indicate that a lens with congenital nuclear cataract has no synthetic action of vitamin C. Yukihiko Mitsui.

Illig, Karl M. **Damage to the retina in cyanide poisoning.** Klin. Monatsbl. f. Augenh. **120**:310-312, 1952.

Cyanide poisonings are usually quickly lethal. This patient survived for two months after an accidental inhalation of the poisonous gas. A severe nephrosis finally caused death. There was a tem-

porary macular edema after venostasis. Later a pigmentation in the retina developed, probably as a result of blood stasis in the choroid. (3 references)

Frederick C. Blodi.

Niedermeier, Siegfried. **Quantitative fractionated protein determinations in the aqueous.** Klin. Monatsbl. f. Augenh. **120**:644-645, 1952.

The author used the method of paper-electrophoresis. He could show that the normal human aqueous contains 0.013 gm./l protein of which 0.01 gm./l is albumin and 0.003 gm./l globulin. (1 figure, 2 references) Frederick C. Blodi.

Niedermeier, S. **A new miotic.** Klin. Monatsbl. f. Augenh. **120**:410-411, 1952.

Pyridostigmin is a prostigmin derivative with less toxicity and longer duration of its effect. Frederick C. Blodi.

Oeri, Johann Jakob. **The problem of noncoagulability of hemorrhage in the anterior chamber.** Acta ophth. **30**:181-201, 1952.

In a detailed report of an investigation on the aqueous of oxen to determine the factors responsible for the noncoagulation of blood in the anterior chamber, the possible factors are grouped under three headings: 1. the morphology of the wall of the anterior chamber, 2. anticoagulants in the aqueous, and 3. dilution of the blood by the aqueous. The author investigated the comparative recalcification capacity of the ox aqueous, with normal physiologic saline as the control, the recalcification time, the effect of the addition of various dilutions of ox aqueous on coagulation of human blood, Quick's test on various dilutions of ox aqueous, a chemical examination of the aqueous for its heparin content, the behavior of hemorrhage in the anterior chamber of the enucleated ox eye and traumatic hemorrhages in the rabbit eye, and the action of the secondary aqueous. The processes responsible for the noncoagulation of hemorrhages in the anterior chamber were not clarified. Oeri suggests that patients with hyphema be subjected to frequent slitlamp study, and that blood obtained by puncture of the anterior chamber be subjected to tests which would demonstrate whether it is capable of coagulation spontaneously or after the addition of thrombin. (4 tables, 9 references)

Ray K. Daily.

Ogino, S., Yamada, Y., and Ichihara, F. **Biochemical study of naphthaline cataract.** Acta Soc. Ophth. Japan **56**:1281-1285, Nov., 1952.

In rabbits in which naphthaline was administered, ascorbic acid decreases rapidly in the lens, while dehydroascorbic acid and 2-keto-gulonic acid increase. An enormous administration of ascorbic acid does not result in a considerable increase in ascorbic acid in the lens of such rabbits. Yukihiko Mitsui.

Palich-Szanto, Olga. **Toxicity of PAS.** Klin. Monatsbl. f. Augenh. **120**:574-577, 1952.

A generalized exanthem after treatment with paraminosalicyclic acid was thought to be a papulonecrotic form of tuberculosis and dissemination was ascribed to the drug. (6 references)

Frederick C. Blodi.

Pentini, Gianfranco. **Cortisone and the repair of experimental corneal lesions in rabbits.** Gior. ital. oftal. **5**:373-386, Sept.-Oct., 1952.

Local administration of cortisone does not delay healing in superficial experimental corneal lesions. Penetrating corneal wounds showed a delayed healing under local treatment with cortisone or insulin. A combination of cortisone and insulin did not influence the process. (12 figures, 21 references) John J. Stern.

Redi, F., and Toxiri, A. **The effect of eserine on corneal anesthetics.** Gior. ital. oftal. **5**:304-309, July-Aug., 1952.

Eserine in a 1/100,000 solution enhances the anesthetic effect of novocain, farmocain and eucopin on the cornea of rabbits. It is antagonistic to the action of butocain and percain. The mechanism of this action has nothing to do with cholinesterase inhibition and cannot be explained at present. (1 table, 11 references)

John J. Stern.

Reiser, K. A. **Studies on sulfonamid therapy.** Klin. Monatsbl. f. Augenh. **120**: 236-246, 1952.

The rate of excretion of sulfonamides after systemic administration was studied. Most of the drug is excreted with the urine. The rate of excretion depends on the affinity of each drug for blood. The excretion during the first 24 hours varies from 65 to 81.5 percent. (5 figures, 12 references) Frederick C. Blodi.

Reiser, K. A. **Studies on sulfonamid therapy.** Klin. Monatsbl. f. Augenh. **120**: 561-574, 1952.

The author studied the sulfonamid concentration in various tissues and confirmed well-known facts. In one group of organs the concentration of the drug is approximately the same as in the blood (uvea, cornea); in another group the concentration is higher than in the blood (conjunctiva) while a third group has a much lower concentration (lens, vitreous). The relation between the bloodlevel and the concentration in a tissue is a constant one. (2 figures, 20 references)

Frederick C. Blodi.

Remler, O. **Bacteriologic and clinical examinations on the effects of antibiotics on microorganisms pathogenic for the eye.** Klin. Monatsbl. f. Augenh. **120**:578-596, 1952.

The author reports a series of bacteriologic and animal experiments in which he tested the bacteriocidal and bacteriostatic effect of local penicillin applications, but he only considers solutions and ointments. Some mixtures of penicillin with other antibiotics or with sulfonamides were evaluated. (12 figures, 19 references) Frederick C. Blodi.

Rizzini, Vittorio. **Tri-paramino-salicylate of dehydro-streptomycin in experimental tuberculosis of the eye.** Gior. ital. oftal. **5**:354-359, July-Aug., 1952.

The chemical combination of dehydrostreptomycin and P.A.S. was as effective in the treatment of experimental tuberculosis of the eye of rabbits as the use of both drugs separately. (2 figures, 30 references) John J. Stern.

Santoni, A. **Experimental toxoplasmosis infection of the eye of rabbits.** Gior. ital. oftal. **5**:279-292, July-Aug., 1952.

Infectious material from the peritoneal cavity of infected rats was instilled into the conjunctival sac of four rabbits, injected into the anterior chamber of four others and into the vitreous of one. All animals in the second and third group developed typical retinal lesions accompanied by violent inflammatory reactions within a few days. One animal in the first group developed a conjunctivitis and died on the thirteenth day from a generalized infection. The inner eye was not affected, but the organisms were found in the spleen. (12 figures, 10 references)

John J. Stern.

Shimoda, Shigemasa. **Influence of low temperature upon corneal regeneration.** Acta Soc. Ophth. Japan **55**:1216-1238, Dec., 1951, & **56**:1222-1229, Nov., 1952.

A corneal erosion was made in the rabbit cornea. When the rabbits were kept at −30 to −40°C for one to two hours, there was a delay in the epithelial regeneration during the low temperature expo-

sure. After the cessation of the exposure, however, there was a considerable acceleration in the regeneration, which not only covered the previous retardation but also resulted in a shortening of the healing course as a whole. When the exposure was made for three hours, the delay in the regeneration persisted. However, in rabbits which were accustomed to low temperature, such a persistent delay was not observed after exposure for three hours. In a second experiment an interstitial defect was made in the cornea. Similar results were obtained as in the case of corneal erosion.

Yukihiko Mitsui.

Šimková, M., and Zahn, K. **The effect of hydergine on retinal circulation.** Ophthalmologica **124**:39-49, July, 1952.

Hydergine is a mixture of three hydrogenated ergot alkaloids, dihydroergocornine, dihydroergocristine and dihydroergokryptine. The drug, therefore, belongs to the group of adrenergic inhibitors, causing, in doses of .3 mg. subcutaneously, a drop in systolic blood pressure, slowing of the pulse rate and a slight drop in ocular tension. These effects occurred in normal individuals and, to a somewhat greater extent, in individuals with hypertensive cardiovascular disease. The effects of the drug upon the retinal circulation were studied by plotting the physiological angioscotomas just before and again 40 to 50 minutes after the subcutaneous administration of the drug. A significant reduction in the area of the angioscotomas was observed in all cases. "For this reason hydergine provides a means of obtaining a prolonged improvement in retinal blood supply and metabolism not only in normal subjects, but also, and this is far more important, in hypertensive and arteriosclerotic patients." (5 tables, 19 references) Peter C. Kronfeld.

Simonelli, M., and Calamandrei, G. **Investigations on the permeability of the blood-aqueous barrier. IX. Influence of local cortisone administration.** Gior. ital. oftal. **5**:364-372, Sept.-Oct., 1952.

The permeability of the blood-aqueous barrier was examined with fluorescein in 29 persons. Five of them had active iritis or iridocyclitis. After three days of local administration of 1-percent cortisone ointment, the permeability of the blood-aqueous barrier was lowered only in the eyes of normal subjects below the age of 15 years. In inflammatory conditions, this effect of cortisone is most pronounced if the disease is in an acute stage. (2 figures, 14 references) John J. Stern.

Simonelli, M., and Rizzini, V. **Isonicotinic acid hydrazide in ophthalmology.** Gior. ital. oftal. **5**:293-303, July-Aug., 1952.

Subconjunctival injections and conjunctival drops were well tolerated in the eye of human subjects and rabbits. Injections into the aqueous and the vitreous were followed by violent uveal reactions. After isonicotinic acid hydrazide injection of 50 mg., a concentration up to 40 gamma per cc. could be detected in the aqueous. Subconjunctival injection of 50 mg. was followed by an aqueous concentration up to 140 gamma. (3 figures, 25 references) John J. Stern.

Studnitz, G. v. **The increased dark adaption after "Adaptinol."** Klin. Monatsbl. f. Augenh. **120**:632-636, 1952.

In this short article the author defends his Adaptinol against a report by Wuestenberg (Klin. Monatsbl. f. Augenh. 119: 524, 1951) who could not confirm an increase in dark adaption after the administration of this drug. (12 references)

Frederick C. Blodi.

Vilstrup, Grethe. **Does the choroid take part in the formation of the aqueous humor?** Acta ophth. **30**:167-172, 1952.

In rabbits the function of the ciliary

body can be abolished easily by diathermy coagulation of the long posterior ciliary arteries. It is then easy to bring about a complete stop of the circulation in the part of the eye where the aqueous humor is assumed to be formed. By the injection of fluorescein into the rabbit's ear and examination of the aqueous in situ or withdrawn from the anterior chamber, these experiments showed that passage of fluorescein still continues from the blood vessels to the aqueous although in reduced quantity. With the circulation of the ciliary body eliminated, it is reasonable to assume that the passage of fluorescein has taken place through the vessels of the choroid. Injection of histamine into the vitreous body was followed by a greater flow of fluid than in the control eye. On the basis of these experiments the author believes that the choriocapillaris participates in the formation of aqueous. Histamine is believed to cause venous stasis, as a result of which resorbtion of fluid is inhibited and ultrafiltration increased. Ray K. Daly.

Vilstrup, Grethe. **Studies on the vascular capacity and tissue fluid content of the choroid and their variation under treatment with histamine.** Acta ophth. **30**:173-180, 1952.

The choroid of rabbits was filled with a plastic mass which was injected into the carotid arteries. As the plastic quickly solidified, the animals were soon dead. The cast formed in the choroid was removed and its weight recorded. Then it was immersed for three days in a concentrated solution of hydrochloric acid which lead to complete dissolution of the tissue elements. The weight of the remaining cast, which represented the vascular content of the choroid, was about one fourth less than the initial weight; apparently three-fourths of the choroidal structure consists of its vessels. In another series of animals histamine was injected into the vitreous of one eye just before the injection of the plastic into the carotid arteries. The effect of the histamine on the fluid content of the eye was then determined by using the second eye of the same animal as a control. The choroid subjected to the action of histamine weighed about four times more than the normal choroid. In spite of the fact that the increase in weight must have been produced by an increase in blood content, the loss in weight due to the subsequent acid corrosion of the tissues was also four times as great as in the control eye. Since the weight of the stroma in both eyes must have been the same, it is concluded that the increased weight of the tissues following histamine injection is caused by an edema of the tissues. (2 figures, 1 table) Ray K. Daily.

Weigelin, E., and Althaus, H. **The retinal arterial pressure during the cold pressor test of Hines and Brown.** Ophthalmologica **124**:1-11, July, 1952.

On 60 subjects, free of circulatory disease, the blood pressures in the brachial and in the central retinal artery were measured during the cold pressor test of Hines and Brown (immersion of one hand in ice water for one minute). The average rise in systolic brachial pressure was 5.8, in diastolic brachial pressure 7.0 mm. The corresponding figures for the pressure rise in the central retinal artery were 5.5 systolic, and 4.5 diastolic. The ratio of the rises in pressure at the two sites, brachial and retinal, suggested the occurrence of an active contraction of the intracranial vessels during the cold pressor test. The authors, apparently, did not try to corroborate this theoretical conclusion by actual measurements. (9 references) Peter C. Kronfeld.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Pascal, J. I. **Lens replacement of accommodative activity.** Ophthalmologica **123**:394-396, June, 1952.

For the accurate calculation of the lens necessary to replace all or part of the normal accommodative activity the author recommends the formula

$$L' = \frac{A}{1-dA}$$

wherein L' is the replacing lens, A the amount of accommodation to be replaced and d the distance between the lens and the first principal plane of the eye in meters. (1 reference)

Peter C. Kronfeld.

Pollice, Franco R. **The reversal of the retinal image. (Why do we see objects correctly and not upside-down?)** Gior. ital. oftal. **5**:435-441, Sept.-Oct., 1952.

The author discards all well-known theories explaining why we do not see objects upside-down and produces his own, which postulates that it is neither due to mental automatism nor to muscular or luminous stimuli. He believes that we mentally project the inverted retinal images into space as in a movie projector. This process of "taking the picture" and of projecting it is simultaneous and does not require cortical awareness of the inverted retinal image. (9 references)

John J. Stern.

Santoni, Armando. **The corneal thickness in cases of high myopia.** Rassegna ital. d'ottal. **21**:219-224, July-Aug., 1952.

Eighty cases of myopia of 10 or more diopters were studied by a special technique to determine the thickness of the cornea. The results were compared to the findings in 80 emmetropic eyes. The thickness of the cornea in the former was 0.012 mm. less than the latter. The method of study consisted of the calculation of the corneal thickness between two foci of light, one falling on the anterior surface and the other on the posterior face of the tissue. (2 figures, 4 references)

Eugene M. Blake.

Sato, T., Shibata, H., and Akiyama, K. **Surgery of myopia, incision of posterior corneal surface.** Acta Soc. Ophth. Japan **56**:1137-1140, Oct., 1952.

Radiating, peri-pupillary incisions of the posterior corneal surface result in a flattening of the cornea, therefore, in a reduction of the degree of myopia. Sato designed a special knife for this operation. After a series of animal experiments, the authors tried the procedure in 30 human myopic eyes. An average reduction of three diopters was obtained. Visual acuity without glasses was improved in all cases. No unpleasant complications have been observed. Yukihiko Mitsui.

Schubert, G., and Bornschein, H. **The human electroretinogram.** Ophthalmologica **123**:396-413, June, 1952.

A quantitative study of the human electroretinogram (ERG) was made on 14 normal subjects and on four representatives of typical congenital visual defects, namely two protanopes, one deuteranope and one person with congenital night blindness. Intensity and spectral composition of the stimulus and the state of accommodation of the patient were varied systematically. Thus the authors obtained ERGs whose quantitatively measurable features, that is the a, b and x-waves could be correlated with distinct visual functions. The authors' method has decided advantages over the standard technique (white light of average intensity) which yields a relatively inexpressive ERG, dominated by just one retinal function, rod vision. (6 figures, 35 references)

Peter C. Kronfeld.

Toselli, C. **Aniseiconia.** Boll. d'ocul. **31**: 463-492, Aug., 1952.

A comprehensive review of the literature is presented. (7 figures, 1 table, 127 references) K. W. Ascher.

5

DIAGNOSIS AND THERAPY

Bissell, G. W. **New concepts in the use of ACTH in ophthalmology and otolaryn-**

gology. Tr. Am. Acad. Ophth. **56**:450-456, May-June, 1952.

Under stimulation of ACTH the adrenal cortex increases the production of all its hormones, which can be grouped into three large categories: 1. the androgenic steroids as reflected by the increased excretion of 17 ketosteroids in the urine; 2. the salt and water steroids as reflected by sodium retention and potassium excretion; 3. the sugar or Compound E and Compound F hormones as reflected by increased corticoid excretion, increased excretion of uric acid, and a lysis of the circulating eosinophils which makes possible a clinical estimation of the degree of adrenal stimulation.

Numerous ocular conditions due to inflammatory disease may be cured or modified. In general, the more acute oculopathies are more amenable to permanent cure, whereas chronic disease may only be modified and demand prolonged therapy. It should be remembered that because of the masking of the usually expected inflammatory reactions by ACTH, underlying infection may not be manifest and, therefore, adjunctive antibiotic therapy should be given, especially to patients receiving ACTH for prolonged periods.

ACTH may be given in a dose of 25 to 50 mg. intramuscularly every six hours around the clock; or 20 mg. in 500 cc. of 5 percent glucose in distilled water intravenously over an eight hour period. A long-acting preparation in gelatin, often necessitating only one injection daily, is now available. Cortisone may be given intramuscularly or by mouth. In ocular conditions it may also be administered topically or by subconjunctival injection. ACTH has no topical application. The clinical usefulness of ACTH and cortisone is due to their effects in altering tissue reaction to hypersensitivity and inflammation. It should be remembered that once fibroblastic proliferation, collagen formation or degeneration has occurred, regeneration of normal tissue should not be expected. Intravenous ACTH is the most potent of the hormonal weapons now available. Theodore M. Shapira.

De Gennaro, Guiseppe. **"Acqua rosa of Castellemare" in ophthalmologic therapy.** Rassegna ital. d'ottal. **21**:289-305, July-Aug., 1952.

The author praises the efficacy of rose water for conjunctival inflammation. The solution contains carbonic anhydride, chlorides of sodium, potassium and magnesium, bicarbonates of sodium and calcium, iron hydrate and calcium sulphate. The fluid remains clear indefinitely.

Eugene M. Blake.

Mariotti, Lorenzo. **The therapeutic uses of the contact lens.** Rassegna. ital. d'ottal. **21**:254-262, July-Aug., 1952.

The many pathologic conditions for which the application of the contact lens has been tried are well reviewed. The author then describes nine cases in which the contact lens afforded definite help, namely, diffuse disepithelialization of the cornea, chemical burns, recent infective keratitis, symblepharon, herpetic keratitis and over-correction of ptosis. (1 figure, 8 references) Eugene M. Blake.

Miller, Henri A. **The dangers of penicillin in ophthalmology.** Ann. d'ocul. **185**: 738-743, Aug., 1952.

Penicillin is potentially dangerous if improperly used. The first potential danger is hypersensitivity. Approximately 5 percent of 1,000 patients treated with penicillin for ocular diseases were hypersensitive to the drug. A patch test is frequently of value in determining the degree of hypersensitivity. The second danger lies in its use in infective diseases which do not react to penicillin; the last danger in dosage and method of administration. Topical administration in

diseases of the eye is frequently unsatisfactory because of the medium used. Obviously, treatment with penicillin should begin at the earliest practical stage of the disease and with bacteriacidal concentrations. Chas. A. Bahn.

Mueller, Fritz. **The importance of the sella bridge for the eye.** Klin. Monatsbl. f. Augenh. **120**:298-302, 1952.

The author reviewed more than 1,000 X-ray films and found a bridge or a pseudobridge in 7 percent of the cases. No definite correlation of these radiologic findings to the optic nerves or its vessels could be found. (8 references)

Frederick C. Blodi.

Sano, S. **The use of Middlebrook-Dubos reaction in ophthalmology.** Acta Soc. Ophth. Japan **56**:1273-1277, Nov., 1952.

This is a study on the specific agglutination reaction of erythrocytes which had been sensitized with tuberculin, after contact with aqueous humor taken from eyes with tuberculosis. The reaction was strongly positive in patients with a tuberculous lesion in the anterior segment of the eye, but was apt to be negative in tuberculosis of the posterior segment.

Yukihiko Mitsui.

Simonelli, M., and Rizzini, V. **The ganglioplegic drugs in ophthalmology.** Gior. ital. oftal. **5**:310-313, July-Aug., 1952.

Pendiomid (Ciba), a pentamethonium compound, was administered parenterally to normal subjects and to a number of patients with various eye conditions. A slight constriction of the pupil and paresis of accommodation was usually observed. In normal subjects the pressure of the retinal arteries and the ocular tension were slightly lowered. In glaucoma (7 chronic non-congestive, 4 acute, 3 secondary), both blood pressure and ocular tension fell considerably after 30 minutes and stayed lowered for several hours. In one case of acute glaucoma, the tension could be lowered from over 100 mm. Hg to normal with retrobulbar and intramuscular injections every 7 to 10 hours. In occlusion of the central retinal artery the drug failed to influence the condition. In two cases of herpes zoster ophthalmicus a marked analgesic effect of the drug was noted. (1 figure.) John J. Stern.

Suda, K., and Kaneda, S. **A new electric manometer.** Acta Soc. Ophth. Japan **56**:1269-1272, Nov., 1952.

The authors introduce a new self-registering electric manometer designed by themselves. A detailed design and an example of respiratory and pulsatory waves of the ocular tension measured by the instrument are illustrated.

Yukihiko Mitsui.

Treumer, Karlheinz, **Tissue therapy in eye diseases.** Klin. Monatsbl. f. Augenh. **120**:624-632, 1952.

Of 45 patients who were given placental extracts, 24 with degenerative affections (myopia, degeneration of the macula, retinitis pigmentosa) showed considerable improvement. (41 references)

Frederick C. Blodi.

6

OCULAR MOTILITY

Guskov, M. I. **A case of voluntary nystagmus.** Vestnik Oftal. **29**:45-46, Jan.-Feb., 1950.

The author describes a case of a young man perfectly normal in every respect who can produce horizontal nystagmus voluntarily. This nystagmus was pendular, rhythmic, not affected by direction of the gaze. It could be maintained for about four minutes. If maintained longer a headache would be caused. Possibility of a voluntary nystagmus should be kept in mind where malingering is suspected.

Sylvan Brandon.

Latte, B. **Unusual combination of abduction insufficiency and a rare familial elbow dysgenesis.** Boll. d'ocul. **31**:491-495, Aug., 1952.

Four of nine siblings showed a marked inability to extend their arms, the bony impediment being located in the elbow joint. One of these children, the seventh, a girl, in addition to the articular deformity (4 X-ray photographs) had a congenital paresis of the right external rectus muscle without retraction and normal visual acuity in both eyes. The combination seems to be described here for the first time. Abducens paresis has been observed in combination with pes equinovarus. (3 figures, 15 references)

K. W. Ascher.

Sebas, S. Raphael. **Heterophorias in aviation medicine.** Rev. brasil. oftal. **9**: 159-164, March, 1951.

The author reviews the phorias, pointing out the possibility of serious and even fatal accidents in aviation due to faulty muscle balance at crucial moments. He believes that the eyes should be tested under as diverse conditions as possible and that the phorias should be tested for when the eyes are tired, thus making certain that men selected for pilot training have no ocular imbalance to interfere with, or prevent, the accomplishing of their mission. (12 references)

Theodore Bisland.

7

CONJUNCTIVA, CORNEA, SCLERA

Bengisu, Noci. **Present status of medical and surgical therapy in trachoma.** Rev. internat. du trachome **29**:87-143, 1952.

The sulfonamides and the antibiotics, administered locally or systemically, do not bring about a definitive cure of trachoma, although they do ameliorate subjective symptoms, diminish conjunctival and corneal vascularization and reduce granulations. Antibiotic ointments are generally the therapy of choice, but silver nitrate and copper sulphate are still of occasional value. (2 figures, 119 references)

James E. Lebensohn.

Borsello, Guiseppe. **Familial reticular dystrophy of the cornea.** Rassegna ital. d'ottal. **21**:272-289, July-Aug., 1952.

The author gives a full bibliographic review of familial reticular dystrophy. He then describes two cases, a father and son, affected by the same form, bilateral in the father and unilateral in the son. The clinical picture is characterized by striae of glassy appearance disposed in lines extending from the limbus and running in the superficial layers of the stroma. Both patients reported recurrent, violent attacks of inflammation, with extensive loss of epithelium and headache associated with acute respiratory infections. The pathogenesis is believed to be a physico-chemical disturbance of corneal tissue provoked by a pseudo-allergic reaction superimposed on a constitutional predisposition. (2 figures, 93 references)

Eugene M. Blake.

Brand, I. **Metastatic furunculiform episcleritis.** Klin. Monatsbl. f. Augenh. **120**:393-396, 1952.

A case of metastatic episcleritis without perforation of the globe is described.

Frederick C. Blodi.

Contino, F. **Ocular manifestations during the course of Stevens Johnson disease.** Riv. ital. del trac. e di pat. ocul. esot. **4**: 5-17, Jan.-March, 1952.

A case of Stevens Johnson disease, extensively studied on the dermatologic service, is reviewed in all its varied manifestations. The ocular findings were notable; the palpebral conjunctiva showed a follicular change in the right eye, in which there was an elevated plaque resembling granulation tissue, and the left

eye showed gray perilimbal elevations suggestive of phlyctenules. Histologic examination and implantation of tissue into the anterior chamber of the rabbit suggested a viral origin. (4 figures, 78 references) Francis P. Guida.

Friede, Reinhard. **Chronic conjunctivitis caused by incarceration of the caruncle.** Klin. Monatsbl. f. Augenh. **120**:538-539, 1952.

In a patient with slight exophthalmus the closing of the lids caused an incarceration of the caruncles. This caused a stasis with chronic irritation which was relieved by excision of the caruncles.

Frederick C. Blodi.

Geserick, H. **Conjunctivitis caused by the hair of caterpillars.** Klin. Monatsbl. f. Augenh. **120**:373-393, 1952.

Within a short period of time 18 patients with conjunctivitis caused by the hair of Dendrolimus pini were seen. The author made careful and important observations. The hair may not be visible in the beginning, but the eye is irritated, there is a local thickening in the conjunctiva and the linear scratches in the corneal epithelium are characteristic. A few days later the hair is often visible and can be extracted with a toothless forceps. These hairs have a great tendency to wander. They enter the cornea at the limbus, coming from the conjunctiva. In the cornea they wander, always surrounded by an infiltrate, until they reach the anterior or posterior surface. A direct, forceful perforation of the cornea, which was assumed to be the cause of the real ophthalmia nodosa, was never observed by the author. The hairs wander because they have a hook on their sharp end and because they are propelled by the cellular reaction around them.

Frederick C. Blodi.

Guenther. **The treatment of xerosis after pemphigus.** Klin. Monatsbl. f. Augenh. **120**:306-310, 1952.

The dryness of the eye after an attack of pemphigus is one of the most intractable complications but the author achieved an excellent result in a patient with severe changes. After mucous membrane transplantations a moist chamber was applied most of the time. The graft took and the keratinized cornea became normal again after 30 years. Vision improved considerably. (4 figures) Frederick C. Blodi.

Illig, K. M. **Digestive enzymes for the treatment of corneal diseases.** Klin. Monatsbl. f. Augenh. **120**:403-407, 1952.

The author used an ointment containing pancreatic enzymes in 37 patients with various corneal diseases and achieved good results. Frederick C. Blodi.

Kornilova, A. F. **Two cases of penetration of larvae of Oestrum ovi into the conjunctival sac.** Vestnik Oftal. **29**:41-42, July-Aug., 1950.

The author describes two cases of infestation of the conjunctival sac with the larvae of the Oestrum ovi. The patients complained of congestion and feeling of swelling in the eye. The larvae were mechanically removed. The eyes cleared. A drawing of the larvae of the Oestrum ovi is presented. Sylvan Brandon.

MacCallan, A. F. **The signs and treatment of trachoma.** Rev. internat. du trachome **29**:235-251, 1952.

The juxtatarsal tissue and the tarsus as well as the conjunctiva are affected in most cases. Fibroblastic proliferation is the most striking change. The Meibomian glands often undergo partial or complete disappearance with replacement by scar tissue. Entropion results from cicatrical contraction of juxtatarsal tissue. Once the tarsus has become involved the application of drugs is useless and surgical

therapy is indicated. (7 figures)

James E. Lebensohn.

Marconcini, E. **A case of perforating scleritis.** Gior. ital. oftal. 5:442-451, Sept.-Oct., 1952.

A man, 72 years old, had ulceration of the bulbar conjunctiva at 9 o'clock near the limbus. The tissue was excised and covered with a conjunctival flap. Three weeks later it had healed. Eleven months later the same condition, with perforation and iris prolapse, was treated by iridectomy and conjunctival repair. After six months, a second relapse at the same site necessitated enucleation. Histologic examination did not explain the etiology of this condition. The author suggests the term nodular perforating scleritis to differentiate it from van der Hoeve's scleromalacia and porphyrin scleromalacia. (2 figures, 44 references) John J. Stern.

Monciino, N. **A statistical study of trachoma in the city of Catania for the last 15 years.** Riv. ital. del trac. e di pat. ocul. esot. 4:31-40, Jan.-March, 1952.

The study revealed a marked reduction in trachoma in the postwar years. Most of the new cases appear in the spring, summer, and autumn. The greatest amount of injury occurs in patients 16 to 30 years of age. The female is the more frequently affected. The disease appears more often in poorer individuals living in close, unhealthy quarters, and who neglect hygiene. The great decrease in the number of trachoma cases can only be attributed to the modern forms of medical treatment. (4 tables)

Francis P. Guida.

Perris, Francesco. **The activity of the Ente Provinciale Antitracomatoso from 1939-40 to 1951-2 and the results of the struggles against trachoma in the province of Cosenza.** Riv. ital. del trac. e di pat. ocul. esot. 4:23-29, Jan.-March, 1952.

The author reviews the history and the manifestation of trachoma, and then shows the activity of the EPA in the province of Cosenza. The brilliant result of its work is noted. Over 60 percent of the patients were cured. The remaining 40 percent included patients who discontinued treatment or used it irregularly. The hope is expressed that such social assistance will be continued and produce better and better results.

Francis P. Guida.

Pletneva, N. A., and Stiksova, V. N. **Oculoglandular form of listerellosis.** Vestnik Oftal. 29:17-21, July-Aug., 1950.

The author describes nine cases of the oculoglandular form of listerellosis. The symptoms were congestion and infiltration in the fornices of one eye, large vascularized follicles, and a granuloma about 5 mm. in diameter. The bulbar conjunctiva and the cornea were clear. There was a marked periauricular, maxillary, cervical and axillary lymphadenitis. Four of the patients had fever up to 40°C. In four cases there was lymphocytosis, in one eosinophilia. Because of similarity to tularemia, tests were made for it, with negative results. Inoculation of a guinea pig produced serum which gave a positive reaction in the Paul-Bunnell test.

Sylvan Brandon.

Ricci, Augusto. **Chloramphenicol, penicillin and sulfonamides in the treatment of trachoma.** Riv. ital. del trac. e di pat. ocul. esot. 4:19-22, Jan.-March, 1952.

Chloramphenicol in one-percent ointment proved to be less effective than penicillin-sulfonamide ointment applied locally. There were no cases of sensitization to chloramphenicol. It is possible that the one-percent ointment is too weak and further tests are projected.

Francis P. Guida.

Rumpelhardt, K. **The relationship be-**

tween myopia and corneal scors. Klin. Monatsbl. f. Augenh. **120**:397-403, 1952.

In evaluating 12,000 cases of myopia and more than 5,000 patients with corneal scars the author confirms the finding that corneal scars are often associated with myopia. Frederick C. Blodi.

Sørensen, C. H. Ravn. **Obstetric lesions of the cornea with formation of hyaline cords in the anterior chamber.** Acta ophth. **30**:223-227, 1952.

In the case reported the lesion occurred in the patient's right eye, and consisted of six vertical cords originating above almost at the limbus and passing downward and temporally, and of three small ruptures of Descemet's membrane. The cords were on the posterior corneal surface, except those situated most temporally which in part were floating freely in the anterior chamber. (1 figure, 23 references)

Ray K. Daily.

Victoria, Virgilio. **Results of trachoma therapy with sulfonamides and antibiotics.** Rev. internat. du trachome **29**:206-234, 1952.

The Trachoma International Prize of 100,000 francs was unanimously awarded to this Argentinian investigator for the best paper on this assigned subject. The author maintains that clinical examination is of greater diagnostic value than microbiological testing. Trachoma is not cured by the sulfonamides even after prolonged use, but they give more rapid relief than any other method introduced before 1937. The administration of sulfonamides has the signal advantages of low cost, stability and mildness. Penicillin does not cure trachoma but adds to the efficiency of sulfa medication; the combined use of penicillin and sulfa is indicated at the beginning of treatment. Aureomycin, if given both locally and orally, yields the most favorable results, and in only a few days has cured patients. Its high cost is the disadvantage from the medico-sociological view. Therapeutic results with streptomycin, chloromycetin and terramycin have been dubious. (13 references)

James E. Lebensohn.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Denig, Rudolf. **The importance of cortisone, ACTH, and animal experiments for my theory on sympathetic ophthalmia.** Klin. Monatsbl. f. Augenh. **120**:312-314, 1952.

The author discusses the role of animal experiments for his theory on sympathetic ophthalmia, which he calls a psychochemostatic inflammation.

Frederick C. Blodi.

Filshtinskaia, L. S. **A case of sympathetic ophthalmia cured with antibiotics and sulfidin.** Vestnik Oftal. **29**:40-41, Nov.-Dec., 1950.

A case of sympathetic ophthalmia is described in which the exciting eye was enucleated because of severe injury and uveitis. In the sympathizing eye the inflammation was severe enough to bring the vision of the eye down to 0.3. Treatment with penicillin, streptomycin and sulfidin was given in subconjunctival injections. After a few weeks of treatment all congestion and precipitates disappeared. Vision improved to 1.0. The total amount of penicillin used was 12 million units, of streptomycin 950,000 units. The author ascribes the cure to the action of streptomycin. Sylvan Brandon.

Purper, H. **Tuberculous uveitis and pregnancy.** Klin. Monatsbl. f. Augenh. **120**:302-306, 1952.

This paper is based on the observation of 35 pregnant women with tuberculous uveitis. Eleven of these were examined by the author himself. Like pulmonary tuberculosis, the uveitis gets worse especially

during puerperium and lactation. An interruption of the pregnancy may be indicated. (12 references)

Frederick C. Blodi.

9

GLAUCOMA AND OCULAR TENSION

Alajmo, B., and Simonelli, M. **Can keratoconus be the clinical equivalent of glaucomatous cupping of the optic papilla?** Gior. ital. oftal. **5**:361-363, Sept.-Oct., 1952.

In three cases of keratoconus the intraocular tension rose after flattening of the cornea with diathermic coagulation. The author discusses the question whether keratoconus might mask latent glaucoma.

John J. Stern.

Batarchukov, R. A. **Observation on the clinical course of glaucoma in hypertensives.** Vestnik Oftal. **29**:3-8, Jan.-Feb., 1950.

The author reports his observation on 730 patients with primary glaucoma, 271 of whom had hypertension. There was no direct relation in the changes of the two pressures in their 24 hour curves, however, some may exist despite the fact that the etiology of neither one is definitely established. Both hypertension and glaucoma have central nervous and vascular components. Out of 271 hypertensives 178 were women, 154 cases were between 61 and 70 years of age, and 83 between 51 and 60. In 111 hypertensives glaucoma was in an advanced stage. Hypertensive changes of the retina were found only in 1.1 percent of glaucomatous patients, whereas in hypertensives without glaucoma it occurred in 77 to 85 percent. When the tension of the eye rises above the level of the half of the diastolic pressure of the brachial artery the retinal circulation suffers. The author uses the term "equilibrium of the retinal nourishment," which is positive when the tension is less than the half of the diastolic blood pressure and negative when it is greater. The equilibrium was positive in 78 percent of the glaucomatous hypertensives and negative in 49 percent. Hypertensives develop glaucoma later than people with normal blood pressure. The majority of patients operated on for glaucoma had negative equilibrium which became positive if the treatment was successful. The author suggests that determination of the equilibrium of the retinal nutrition may be of help in deciding whether to operate or not.

Sylvan Brandon.

Böke, W. **Allergy and intraocular pressure.** Klin. Monatsbl. f. Augenh. **120**:488-503, 1952.

In order to study a possible connection between allergy and intraocular pressure, four patients were examined during an allergic phase. In none of them did the pressure rise beyond normal values. Another patient with chronic glaucoma and known allergy to milk had a marked decrease in the intraocular pressure during the allergic phase. (5 figures, 16 references)

Frederick C. Blodi.

Dubois-Poulsen, A. **The visual fields in chronic glaucoma.** Ann. d'ocul. **185**:673-700, Aug., 1952.

Enlargement of the normal venous angioscotomata, between the upper margin of the disc and the macula, is the initial manifestation of chronic glaucoma. The vessels involved are a part of the circle of Haller-Zinn and therefore are branches of the ciliary and not the central retinal circulation. As the disease progresses these angioscotomata fuse and become more dense, forming arciform scotoma of different shapes (Seidel, Bjerrum). Arciform scotoma may also be produced by numerous diseases ranging from embolism of the central retinal artery to carotid aneurysm in all of which the same receptors and nerve fibres are involved. Glaucoma-

tous defects of the field may be differentiated from similar ones of nonglaucomatous origin because changes follow the instillation of pilocarpine. If defects follow the median raphe, the Rönne step is produced. As contraction increases, a breakthrough occurs into the lower nasal periphery; finally the entire field becomes defective but the macular area usually is lost last. In glaucoma the peripheral isopters retract more rapidly than the central. Field defects in chronic glaucoma usually precede increased ocular tension by years and occasionally the visual fields are completely lost without hypertension. Contractions of the field progress with disc pallor rather than with excavation of the disc. Excavation is primarily the result of anoxemia and of absorption of interstitial tissue in the papilla rather than of shrinkage of nerve fibers (Lacunar degeneration of Schnabel). Visual fields in glaucomatous patients are preferably made with reduced light and with 1/1000 or 1/2000 targets. Color perimetry is of litle practical value. Operations with maximal trauma to the iris (iridectomy) are usually followed by greater field shrinkage than those with minimal iris trauma (cyclodyalysis). Chas. A. Bahn.

Jensen, Siegfried. **Capillary fragility in glaucoma with cataract.** Klin. Monatsbl. f. Augenh. **120**:502-510, 1952.

Capillary fragility was measured by counting the petechial skin hemorrhages caused by suction. The fragility was often lower than normal and gave no clue as to a complication after the operation. (3 figures, 16 references)

Frederick C. Blodi.

Niedermeier, Siegfried. **Dibenamin in inflammatory glaucoma.** Klin. Monatsbl. f. Augenh. **120**:534-538, 1952.

Good, but only temporary, results were obtained in four patients with inflammatory glaucoma. Dibenamin was given orally. In seven of eleven patients with chronic glaucoma there was an additional reduction of the intraocular pressure when they received miotics. (1 figure, 5 references) Frederick C. Blodi.

Posthumus, R. G. **The use and the possibilities of progesterone in the treatment of glaucoma.** Ophthalmologica **124**:17-25, July, 1952.

In a very heterogeneous group of glaucomas the author observed slight to marked lowering of the ocular tension after the subcutaneous or intramuscular implantation of a progesterone preparation called progestine (one pellet contains 100 mg. of the hormone). This effect occurred in both sexes and in primary as well as in secondary glaucomas. The hormone therapy was used in conjunction with miotics and surgery. "As long as glaucoma remains a very complicated abnormality, not yet fully studied, and as long as the hormonal action of progesterone outside the sex organs is not fully understood, we cannot give a clear interpretation of the facts." (6 refernces)

Peter C. Kronfeld.

Prijot, E., and Weekers, R. **Measurement of the resistance to aqueous outflow by means of the electronic tonometer. Part four. The mode of action of pilocarpine in chronic noncongestive glaucoma.** Ophthalmologica **124**:12-16, July, 1952.

In 20 cases of chronic open-angle glaucoma the resistance to aqueous outflow (the reciprocal of Grant's coefficient of facility of outflow) was measured tonographically, first without any medication and then under the influence of pilocarpine. A significant drop in the resistance to aqueous outflow occurred in 17 of the 20 cases. Several different mechanisms may account for this drop in resistance. The authors believe that the drop in outflow resistance results from improved

venous outflow through the scleral plexuses. (15 references)

Peter C. Kronfeld.

Sédan, J., and Sédan-Bauby, S. **The instillation of atropine in absolute glaucoma.** Ann. d'ocul. **185**:734-737, Aug., 1952.

In absolute glaucoma, pain and even ocular tension may be reduced after the instillation of atropine. This is ascribed to an antispasmotic action of belladonna in glaucomatous eyes which are so degenerated that they no longer respond to parasympathetic paralysis. Three illustrative cases are briefly discussed. (4 references)

Chas. A. Bahn.

Treumer, Karlheinz. **The corpus luteum hormone in the treatment of glaucoma.** Klin. Monatsbl. f. Augenh. **120**:523-534, 1952.

Thirty patients with various forms of glaucoma were treated with this hormone. No definite pressure-reducing effect was found. This does, therefore, not corroborate the original claims of Obal. (6 figures, 2 tables, 17 references)

Frederick C. Blodi.

Urrets, A., and Zavalia, H. **Notes on the pathogenesis of glaucoma.** Ann. d'ocul. **185**:701-733, Aug., 1952.

Aqueous is produced in the arteriolar capillaries of the ciliary processes from the blood plasma by processes of ultrafiltration, osmotic pressure and secretion. In both noncongestive and congestive glaucoma the fundamental pathologic process is a specific type of progressive sclerosis which results in anoxemia of the cells and imperfect aqueous re-entry into the venous circulation. In the noncongestive type, which is basic, the venules of the papilla are first involved. These form part of the circle of Haller derived from the uveal circulation. A variable bodily vagotonia or relative parasympathetic deficiency is present which retards the rejoining of the aqueous with the venous circulation. This is illustrated by enlargement of the intraocular, episcleral, and conjunctival veins. Ampulliform excavation of the disc and lacunar atrophy are not caused primarily by increased intraocular pressure, but by the anoxemia which results from sclerotic atrophy of the capillary bed in the papilla. In the basic or noncongestive type of glaucoma the posterior uveal and retinal vessels are primarily involved. In the congestive type the anterior uveal vessels are principally affected; a tendency to plasmoid aqueous results which may be ascribed to a disturbance in the ciliary body. This congestive factor is comparatively labile and is usually modified by autonomic imbalance. The structural pattern of the eye, particularly of the filtration angle, also modifies the congestive reaction. Especially in the noncongestive type ocular tension tends to vary with intravenous pressure and normal daily variations in tension are exaggerated. The classification of glaucoma into narrow and wide angle types, although serving a useful purpose, must not be taken too literally. In the glaucomatous eye, pilocarpine and similar drugs reduce ocular tension by increasing venous capillary dilatation and permeability, thus facilitating the return of aqueous into the venous circulation. Atropine increases ocular tension in the glaucomatous eye by increasing arteriolar permeability and dilatation, thereby interfering with aqueous re-entry into the uveal and retinal venous circulation and indirectly through the canal of Schlemm. (137 references)

Chas. A. Bahn.

10

CRYSTALLINE LENS

Boles-Carenini, B. **Possibility of antihistamine therapy in phacoanaphylactic reactions after cataract operation.** Boll. d'ocul. **31**:449-458, Aug., 1952.

Thirty-one patients in the Parma University eye clinic were given high doses of antihistamine drugs intramuscularly or orally or by instillation into the conjunctiva, in order to combat the irritation deriving from lens cortex remnants after extracapsular cataract operation. The drugs employed were allergin, antadryl, antistine, Dimetin, benzoxal, and pyrobenzamine. They were given on the day of surgery and on the two following days intramuscularly, the following three to seven days by mouth, and twice a day or more often by instillation. A table shows the onset of the reaction, the time when improvement was first noticed and when the reaction disappeared. The latter varied between four and 36 days; the author found the postoperative course shorter in the cases treated in this way. (25 references) K. W. Ascher.

Fedorov, S. A. **Cholesterin in the lens in a cataract.** Vestnik Oftal. **29**:43-44, Jan.-Feb., 1950.

The author discusses the reason of appearance of cholesterin in the eye. A case is presented of a 7-year-old child with congenital cataracts in whom cholesterin crystals were seen in the center of the lens. After the extraction of the cataract the crystaline masses were examined and the presence of cholesterin was confirmed. The mother of the child had cholesterin crystals in her congenital cataracts.

Sylvan Brandon.

11

RETINA AND VITREOUS

Bottoni, Angelo. **Diabetic retinopathy and insulin therapy.** Gior. ital. oftal. **5**:408-422, Sept.-Oct., 1952.

Of 236 patients, 147 showed more or less severe diabetic retinopathy. The percentage is higher in moderately severe diabetes in which balance is maintained with a moderate amount of insulin. Well-controlled treatment with minimum doses of insulin does not provoke diabetic retinopathy. The amount of insulin administered is not related to the incidence of retinopathy, provided that the dosage is commensurate with the patient's need. Insulin neither damages the retina nor is it able to protect it. (2 figures, 1 reference)

John J. Stern.

Fanta, H. **Treatment and success in retinal detachment.** Klin. Monatsbl. f. Augenh. **120**:469-480, 1952.

A review of the records of 202 patients operated on for retinal detachment from 1946 to 1949, at Lindner's clinic in Vienna, shows that the result was good in two-thirds of the cases. (1 table, 22 references)

Frederick C. Blodi.

Friede, Reinhard. **The therapy of congenital retinitis pigmentosa.** Klin. Monatsbl. f. Augenh. **120**:605-624, 1952.

The author reviews some recommended treatments for retinitis pigmentosa. Of 39 patients to whom a combination of vasodilators, milk injections, hormones (follicle hormone, placental extracts), physical therapy and vitamins was given, 15 were improved. A damaged permeability of Bruch's membrane is thought to be the primary cause of retinitis pigmentosa. (2 references) Frederick C. Blodi.

Gasparri, F. **Tromexan treatment of thrombosis of the central retinal vein (results of 25 cases).** Gior. ital. oftal. **5**:395-407, Sept.-Oct., 1952.

Tromexan is the ethyl ester of bis 3-3' (4-oxicumarenyl) acetic acid. It was used in 25 cases of total or branch thrombosis of the central retinal vein, three to 90 days after the onset. Treatment consisted in the injection of 12 mg. initially and 9, 6 and eventually 3 mg. daily for two weeks. In 64 percent of the cases, a good result, with total or partial recovery of vision, was obtained. Side affects like hematuria are mild but the prothrombosin

time must be checked as in dicumarol treatment. John J. Stern.

Nover, A. **A modified marker for detachment operations.** Klin. Monatsbl. f. Augenh. **120**:408-410, 1952.

The curved marker of Amsler was modified to allow its application at the limbus.

Frederick C. Blodi.

Ooka, Yoshiko. **Fundus changes of imbecile children.** Acta Soc. Ophth. Japan **56**:1007-1014, Sept., 1952.

The author found changes in the fundus in 102 (43 percent) of the 235 imbecile children examined. Among them a degenerative change in the macular region which resembles that of hereditary degeneration of the macula was found in 52 cases. Yukihiko Mitsui.

Rebello, Orlando. **Retinitis pigmentosa and tissue therapy.** Rev. brasil. oftal. **9**:121-155, March, 1951.

The author briefly reviews the history of heterogeneous transplantation of tissue with especial reference to the use of this method in the treatment of retinitis pigmentosa. The various other approaches to the treatment of this disease are also presented. The author's experiences are only with subcutaneous and subconjunctival implantation of placenta, and with subcutaneous, subconjunctival, and retrobulbar injection of placental extract. The technique of preparation is given. In addition, a detailed analysis of the chemical composition and especially the amino acid content of the placenta is given, based on work at the Instituto Biochemico Maragliano in Rio de Janeiro. (44 references)

Theodore Bisland.

Tosi, B., and Gayoso, C. B. **Toxic retinitis of Leber.** Arch. oftal. Buenos Aires. **27**:132-135, March, 1952.

In 1909 Leber described under the name "retinitis pseudonephritica stellata" an ophthalmoscopic picture which included exudates, hemorrhages, blurring of the margin of the disc, and the formation of a macular star. The author presents two patients (an 8-year-old boy and a 39-year-old woman) both of whom show the picture of Leber's pseudo-nephritic retinitis with very poor vision. Clinical and laboratory tests for renal involvement were negative. The end result was a moderate post-neuritic atrophy with useful vision in at least one eye. The authors list many hypotheses regarding the genesis of the macular star, and discount strongly the views of some French authors that there must be some renal involvement, though to a degree not shown by the available tests. All the records of these cases show that the prognosis as to life is excellent and that there is spontaneous cure and some improvement of vision.

Joseph I. Pascal.

Wadsworth, J. A. C., Schepens, C. L., Pischel, D. K., Kronfeld, P. C., and Hughes, W. F., Jr. **Symposium: Retinal detachment.** Tr. Am. Acad. Ophth. **56**:370-448, May-June, 1952.

Wadsworth, J. A. C. **Etiology and pathology.** pp. 370-397.

Certain important pathologic processes pertaining to the detachment of the retina have been shown. The importance of the role played by the vitreous has been demonstrated by microscopic sections and an attempt has been made to show the sequence of events that takes place in the formation of a retinal detachment. The results and pathologic findings have been shown after diathermy application. (55 figures)

Schepens, Charles L. **Diagnostic and prognostic factors as found in preoperative examination.** pp. 398-418.

Early symptoms in most cases are: localized flashes of light, often below and

nasally; a sudden increase in vitreous floaters; and often a vitreous hemorrhage which may obscure fundus details. When the detachment increases, a gray cloud appears in the visual field, and light flashes disappear. Between 40 and 60 percent of all detachments occur in myopic eyes and, in the cases seen by the author, over 20 percent were in aphakic patients. Slitlamp examination may reveal atrophy of the posterior pigment layer of the iris, which often indicates marked degeneration of the extreme periphery of the fundus. In the deep portions of the vitreous, a detachment of the posterior hyaloid membrane may be found, which appears as a tense membrane stretched across the vitreous cavity. In practically all cases of retinal detachment, a field defect may be detected provided one uses small test objects or reduced illumination. Ophthalmoscopy should supply information on the following points: 1. is the retina detached?, 2. extent of the detachment, 3. location of the retinal breaks, 4. status of the macula, and 5. the status of the vitreoretinal adhesions. The author also presents differential diagnosis. (22 colored plates and descriptions)

Pischel, D. K. **Treatment of retinal detachments.** pp. 419-431.

The author describes the methods for the localization of breaks, and gives a description of a typical operative procedure. (15 figures, 6 colored plates)

Kronfeld, P. C. **Postoperative care and complications.** pp. 432-438.

There are several true complications which occur during the postoperative period and which are related to the operation or its effects upon the ocular tissues. A benign form of iridocyclitis follows extensive application of the current to any, but specifically to the anterior, portions of the sclera. Suppurative infections of the interior of the eye after electrocoagulations are extremely rare. A typical but infrequent complication is hemorrhage into the vitreous cavity or into or between the layers of the eyeball wall. The fact that after the operation parts of the retina tend to move toward the choroid while others are still held by vitreous, entails physical stresses that can easily lead to retinal hemorrhages. Bleeding from choroidal vessels may be due to partial sloughing. Hemorrhages into the vitreous cavity may assume very bizarre shapes because of the complicated anatomic conditions prevailing in eyes with retinal detachments.

Massive retraction of the vitreous has been mentioned as a postoperative irregularity but probably represents a complication. The same status has to be accorded to the star-shaped retinal folds which occur postoperatively in different sizes and shapes due to several pathogenetic processes. In some cases it is an intraretinal atrophying process, whereas in others vitreoretinal adhesions or newly formed membranes on the inner retinal surface dominate the picture. While star-shaped folds unquestionably occur in eyes not operated upon, they are more common after operations and are, therefore, possibly a direct or indirect effect of the electro-coagulation. Weve's method of treating such areas with roentgen therapy in the early stages and with electrocoagulation aiming at the foci of connective tissue in later stages has been unsuccessful in our hands. Scleral resection may be considered as a means of bringing the folded retina closer to its matrix, the choroid.

The problem in the treatment of the postoperative irregularities and complications of retinal detachment is the question of whether, when, and how to operate again. Just as during the preoperative work-up, close ophthalmoscopic study will prove most helpful in arriving at the proper decision.

Hughes, W. F., Jr. **Evaluation of results of retinal detachment surgery.** pp. 439-448.

A survey of 375 patients operated on for primary retinal detachment by members of this symposium has emphasized several of the points made in previous discussions. Those factors which adversely affect the prognosis for reattachment of the retina include 1. myopia, 2. peripheral cystic degeneration, 3. the size of the detachment (although 48 percent of total detachments were cured), 4. failure to detect and localize retinal breaks, 5. failure of the retina to fall back in apposition to the choroid, 6. blind and possibly excessive barrage with diathermy current, and 7. sclerotomies and sclerectomies for the evacuation of subretinal fluid. The relatively poorer prognosis in aphakic eyes may be partially explained by their larger detachments, less frequent localization of the breaks, and less extensive flattening during bed rest. The results of 38 scleral resections performed largely on aphakic eyes, or after diathermy had failed, showed 18 percent cures. Visual improvement is often dramatic after surgical reattachment of the retina, even when the macula has been detached. (18 tables)

Theodore M. Shapira.

12

OPTIC NERVE AND CHIASM

Börner, Ruth. **Optic neuritis in ophthalmic herpes zoster.** Klin. Monatsbl. f. Augenh. **120**:636-639, 1952.

A patient with herpes zoster of the ophthalmic nerve had not only a conjunctivitis, keratitis and iritis, but also an optic neuritis which ended in an optic atrophy. (16 references) Frederick C. Blodi.

Yonemura, Taizo. **Flicker test in the diagnosis of nutritional retrobulbar optic neuritis.** Acta Soc. Ophth. Japan **56**:1262-1265. Nov., 1952.

The nutritional chronic retrobulbar optic neuritis is very often met with in Japan and the reduction of vision in this condition is, in some cases, very slight or even negligible. Fifteen cases of this condition, in which the visual acuity was 20/20 or more, were examined by a flicker test. A considerable reduction in the fusing power was observed in all cases. The reduction disappeared, however, when the nutrition was improved.

Yukihiko Mitsui.

13

NEURO-OPHTHALMOLOGY

Dolan, R. A., and Bucy, P. G. **Pain of herpes zoster ophthalmicus.** Arch. Neurol. & Psychiat. **68**:314-317, Sept., 1952.

The case of a 74-year-old man is described. A retrogasserian neurotomy was performed for trigeminal neuralgia, and approximately one year later severe herpes zoster ophthalmicus developed on the same side. This was without pain from the onset and remained so until he was last seen, over a year later. It is believed that this case demonstrates that the trigeminal nerve is the only peripheral nerve structure which is concerned in ophthalmic herpes, the pain associated with the acute form, and probably with postherpetic neuralgia. It shows that the various other pathways, sympathetic, vagal, facial and motor fifth, which have been cited as possible pathways for painful impulses from the face are not concerned with the pain of ophthalmic herpes, and probably not with postherpetic neuralgia. However, it must still be recognized that section of the trigeminal nerve will not relieve these pains, and that, therefore, the nerve probably serves only as a pathway to the central nervous system, and disease in the peripheral nerve is not the cause of the pain once postherpetic neuralgia has developed.

Theodore M. Shapira.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at last three months before the date of occurrence.

DEATHS

Dr. Charles Herndon Haralson, Tulsa, Oklahoma, died October 29, 1952, aged 60 years.

Dr. Louis Levy, Memphis, Tennessee, died October 20, 1952, aged 63 years.

ANNOUNCEMENTS

LANCASTER COURSE

The Lancaster Courses in Ophthalmology sponsored by the Ophthalmic Study Council will be held this year at Colby College, Waterville, Maine, beginning June 22nd and running through September 5th. The usual excellent instructors will be on hand, and the same courses will be given. For further information address: The Ophthalmic Study Council, 243 Charles Street, Boston 14, Massachusetts.

GRANT-IN-AID-AWARDS

Applications for the 1953-1954 grant-in-aid awards of the National Council to Combat Blindness will be considered by the organization's Committee on Research at its fourth annual meeting. Completed applications should be received in the office of the council no later than April 15, 1953. Forms may be obtained by addressing: Secretary, National Council to Combat Blindness, Inc., 1186 Broadway, New York 1, New York.

The council, in accordance with its research program, encourages applications for grants to projects encompassing the scientific principles that are related to the study of sight—both normal and pathologic, including the various branches of biologic, chemical and physical science, and clinical investigative medicine.

Serving on the Committee on Research of the National Council to Combat Blindness are: Dr. Phillips Thygeson, University of California Medical College, San Francisco; Dr. James H. Allen, M.D., Tulane University College of Medicine, New Orleans; Dr. Alson E. Braley, University of Iowa, Iowa City; Dr. Dan M. Gordon, New York Hospital-Cornell Medical Center, New York; Charles Haig, Ph.D., New York Medical College, New York; Dr. Stuart Mudd, University of Pennsylvania, Philadelphia; Dr. Charles A. Perera, College of Physicians and Surgeons, Columbia University, New York; and Dr. Samuel L. Saltzman, New York Medical College, Flower and Fifth Avenue Hospitals, New York.

Grants-in-aid approved by the committee for the period 1952-1953 are as follows:

(Continuation) Retina Foundation, Massachusetts Eye and Ear Infirmary, Dr. Endre A. Balzs, $2,500, "Acid mucopolysaccharides in the vitreous body"; University Hospitals, University of Iowa, Dr. Hermann M. Burian, $5,000, "Study of the electrical response of retina and brain in patients with amblyopia ex anopsia and suppression."

New York Eye and Ear Infirmary, Dr. Bernard Goldberg, and Dr. Bernard Levene, $2,500, "The possible role of hyperestrinism in the etiology of retrolental fibroplasia"; Wills Eye Hospital, Philadelphia, Harry Green, Ph.D., and Dr. Irving H. Leopold, $3,750, "Investigation of the intermediary reactions and the enzyme systems involved in the anaerobic glycosis by the lens"; New York Medical College, Charles Haig, Ph.D., $2,625, "Detection of plane polarized light by the human eye."

Stanford University, San Francisco, Dr. Arthur Jampolsky, $2,500, "Visual characteristics of macular and peripheral retina in amblyopia associated with convergent and divergent deviation"; University of Louisville, School of Medicine, Dr. Arthur H. Keeney, $2,445, "Role of estrogenic and androgenic substances on the retinal vascular pattern in alloxan diabetic rats"; Yale University School of Medicine, Dr. J. Alexander Van Houven, $560, "The treating of artificial corneal burns with various substances releated to adrenalin and its derivatives"; Indiana University Medical Center, Dr. Fred M. Wilson, $1,596, "Norms and techniques of corneal biostandardizations of beta irradiation applicators for use in ophthalmology."

PENNSYLVANIA ANNUAL MEETINGS

The 1953 annual meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology will be held May 21, 22, 23, and 24, 1953, at the Galen Hall Hotel, Wernersville (Reading), Pennsylvania.

The 1954 annual meeting has been scheduled for May 20, 21, 22, and 23, 1954, at the Bedford Springs Hotel, Bedford, Pennsylvania.

OREGON ACADEMY MEETING

The annual spring convention in ophthalmology and otolaryngology will be held in Portland, Oregon, March 23rd through 27th. A fine program has been arranged by the Oregon Academy and the University of Oregon Medical School. Ophthalmology meetings will be held Monday, Tuesday, and Wednesday forenoon; otolaryngology, Wednesday afternoon, Thursday, and Friday.

Guest speakers will be: Dr. Hermann Burian, State University of Iowa Medical School, Iowa City; Dr. Harold Scheie, University of Pennsyl-

vania Medical School, Philadelphia; Dr. Kenneth M. Day, University of Pittsburgh Medical School, Pittsburgh; Dr. Jerome A. Hilger, University of Minnesota Medical School, Minneapolis. Dr. Burian will deliver the fourth John E. Weeks Memorial Lecture in Ophthalmology. For further information address: Dr. David D. DeWeese, 1216 S.W. Yamhill Street, Portland 5, Oregon.

Miscellaneous

Montefiore conference

At the recent clinical conference sponsored by the Department of Ophthalmology, Montefiore Hospital, New York, Dr. Edwin D. Billet spoke on "Nonsurgical management of intermittent exotropia"; Dr. Abraham Schlossman, "Pathology of intraocular foreign bodies"; Dr. Max Chamlin, "Management of ocular complications in neurosurgical conditions." Dr. Samuel Gartner and Dr. Carlton C. Phillips presented "Surgical problems from the eye service."

Magazine of color science

Die Farbe, a new German quarterly devoted to all the technical problems of color science, has been launched with Dr. Manfred Richter, editor, whose address is Unter den Eichen 87, Berlin-Dahlem. Representing ophthalmology on the editorial staff are Professor Eggert of Zurich and Professor Engelking of Heidelberg.

Research Study Club

The Research Study Club of Los Angeles arranged the following ophthalmic program for its 22nd annual midwinter postgraduate clinical convention:

Dr. Ramon Castroviejo, New York, lectured on: "Keratectomies and conjunctival plasties," "Keratoplasties," "Discussion of results of keratectomies and keratoplasties," "Treatment of glaucoma by cyclodiathermy," "Cataract surgery," "Nasal pathology and ocular complications," and "Ocular surgery: Miscellaneous."

Dr. Alfred E. Maumenee, Stanford University School of Medicine, San Francisco, spoke on: "What do we know about the etiology of uveitis?"; "Some recent observations on glaucoma," and "Diseases of the conjunctiva and cornea."

Other speakers were: Dr. Watson Gailey, Bloomington, Illinois, "Cataract surgery"; Dr. Harold F. Whalman, Los Angeles, "Developmental anomalies of the human eye"; Dr. I. G. Sommers, Los Angeles, "A comparison of biomicroscopy and histopathology of the eye"; Dr. Warren A. Wilson, Los Angeles, "Management of congenital cataract"; Dr. S. Rodman Irvine, Beverly Hills, "Management of vitreous complications following intraocular surgery"; Dr. Robert J. Schillinger, Los Angeles, "Intravenous nembutal anesthesia in eye surgery."

In addition to these lectures instruction courses were presented by: Dr. Deane C. Hartman, "Fornix and socket reconstruction"; Dr. Wendell C. Irvine, "Clinical pathology and management of epithelial downgrowth"; Dr. George P. Landegger, "Postoperative care of the cataract patient"; Dr. Roderic P. O'Connor, "Up to date on the cinch operation"; Dr. George S. Zugsmith, "Educating the layman in problems of eye care."

A special course in eye surgery was given by Dr. Meyer Wiener and associates, Dr. Maurice W. Nugent, Dr. Clarence H. Albaugh, Dr. A. Ray Irvine, Jr., Dr. S. Rodman Irvine, Dr. Stephen J. Popvich, Dr. Walter L. Roberts, Dr. Carrol L. Weeks, and Dr. Warren A. Wilson.

On the motion picture program were the following films: "The development of the human eye," American Academy production; "Cataract extraction," Dr. Conrad Berens, New York; "Air goniotomy," Dr. A. E. Maumenee, San Francisco; "Hemangioma of the conjunctiva: Removal with pedicle graft for repair," Dr. Wendell L. Hughes, Hempstead, New York; "Lamellar resection of the sclera for retinal detachment," Dr. Milton L. Berliner, New York; "Therapeutic lamellar corneal graft for the treatment of herpetic or bullous keratitis," Dr. E. Townley Paton, New York; "Cataract extraction with three track sutures," Dr. Brendan D. Leahey, Lowell, Massachusetts; "An orbital implant of plastic sponge," Dr. Maurice D. Pearlman, Chicago; "An improved motility implant," Dr. Max Fine, San Francisco; "Pterygium operation: Arlt's technique," and "Motais operation for ptosis," Dr. George P. Landegger, Los Angeles.

Memphis program

At the recent annual convention of the Memphis Eye, Ear, Nose, and Throat Society, Dr. C. Wilber Rucker, Rochester, Minnesota, spoke on "Perimetry," "Clinical classification of nystagmus," and "Diagnostic problems in neuro-ophthalmology"; Dr. A. D. Ruedemann, Detroit, "Foveal coördination: Interpretations of end results," "Exophthalmos: Use of tracer studies and treatment with radioactive iodine," and "The use of X-ray, gamma and beta irradiation in ophthalmology"; Dr. Willis S. Knighton, New York, "Glaucoma: Classification and diagnosis," "Glaucoma: Medical treatment," and "Glaucoma: Surgical treatment."

Speaking at the annual dinner, Dr. Ruedemann described "The American Academy of Ophthalmology and Otolaryngology: Its problems of interest to the profession."

East-Central Research meeting

Dr. Arthur Culler, Columbus, Ohio, was the presiding officer of the recent meeting of the East-Central Section of the Association for Research in Ophthalmology at Columbus. The following papers were presented.

"A method of continuous recording of changes in pupil size," M. J. Allen, Ph.D., Ohio State University, Columbus; "Influence of varidase on lime burns of the eye," Dr. Jay G. Linn, Jr., Pittsburgh; "Study of compensation-maximum test on aqueous veins," Dr. John C. Fuhs, Dr. Joseph L. Stambaugh, Jr., and Dr. Edward J. Glaser, University of Cincinnati, Cincinnati.

"Studies of the visual toxicity of methanol: III. Metabolism of C_{14} methanol in experimental ani-

mals," Dr. Albert M. Potts and Vera Glocklin, M.S., Western Reserve University, Cleveland; "The effect of intracarotid injection of a basic dye on the ciliary body," Dr. E. J. Ballintine and Dr. Lawrence Peters, Western Reserve University; "An experimental study of the broad spectrum antibiotics in herpes simplex encephalitis in mice," Dr. G. A. Thomson, University of Toronto, Toronto.

"Report on study of normal and pathologic nystagmus with the electro-encephalograph," Dr. Donald J. Lyle, University of Cincinnati, Cincinnati; "The occurrence of 'core cells' in the optic nerve of various reptilian species," J. H. Prince, F.R.M.S., Ohio State University; "A new method for measuring binocular fusion compulsion," Dr. Gerhard A. Brecher, Western Reserve University; "Summary of 15 years of research on accommodation and convergence," Glenn A. Fry, Ph.D., Ohio State University; "Temporary disturbances of pituitary-adrenal balance when corticotropin is withdrawn after its prolonged use in eye disease," Dr. J. R. Quinn and Dr. W. Q. Wolfson, University of Michigan, Ann Arbor; "Preliminary studies of variations in spectral sensitivity within the central fovea," H. Richard Blackwell, Ph.D., University of Michigan; "Endothelial dystrophy," Dr. Harry B. McGree and Dr. Harold F. Falls, University of Michigan.

Societies

Irish society to meet

The annual meeting of the Irish Ophthalmological Society will be held at the Royal Victoria Eye and Ear Hospital, Dublin, on April 16th and 17th.

Oto-Neuro-Ophthalmology congress

The XVIII International Congress of Oto-Neuro-Ophthalmology will be held in Bologna, Italy, May 3rd to 7th. The official subjects to be discussed are "Dysraphia in oto-neuro-ophthalmology," and "Meniérè's disease." Prof. Q. Di Marzio is chairman of the congress.

Brooklyn meeting

At the 122nd regular meeting of the Brooklyn Ophthalmological Society, Dr. Alfred Weintraub spoke on "Ophthalmoscopy." Dr. Max Chamlin presented a paper on "Field changes in neurological lesions: Before and after," and Dr. Everett W. Corradini discussed "Ocular disturbances in neurosurgical conditions."

Officers for Cuban society

Officers of the Cuban Ophthalmological Society for 1952-1953 are: President, Dr. Gilberto Cepero Garcia; vice-president, Dr. Manuel Anton; second vice-president, Dr. Alberto de Jongh; secretary, Dr. Jose Alfonso, Jr.; vice secretary, Dr. Guillermo de la Torre Ordetx; treasurer, Dr. Mario Rodriguez Perez, and vice treasurer, Dr. Pedro Sanchez Borroto.

Madrid officers

New officers for the Madrid Ophthalmological Society are: President, Dr. Manuel Marin-Amat; vice-president, Dr. Juan Arjona Trapote; secretary, Dr. Fermin Galindez Iglesias; treasurer, Dr. Manuel Rios Sasiain; director, Dr. Rafael Bartolozzi Sanchez.

Gifford memorial lecture

The ninth annual Sanford Robinson Gifford Memorial Lecture sponsored by the Chicago Ophthalmological Society was delivered by Dr. Frank B. Walsh, Baltimore, on February 14th at the Sheraton Hotel, Chicago. The subject of Dr. Walsh's address was "Some aspects of calcification in ophthalmology."

New York Eye and Ear Infirmary meeting

The Alumni Association of The New York Eye and Ear Infirmary will hold its annual scientific program from April 20th through April 24th. The scientific sessions in ophthalmology will be held Monday and Tuesday; ear, nose and throat on Thursday and Friday at The New York Academy of Medicine, Room 440. Wednesday will be left open for clinical and surgical demonstrations. Morning sessions will commence at 9:30; afternoon sessions at 2:00.

Basic course in orthoptics

The basic course in orthoptics sponsored by the American Orthoptic Council will be given in 1953 in the Department of Ophthalmology, College of Medicine, State University of Iowa, Iowa City, Iowa.

The course will consist of didactic lectures and practical demonstrations, given by outstanding teachers. It will be held from June 15, 1953, through August 15, 1953.

High school graduates are eligible to take this course, but preference will be given to applicants with at least two years of College training, or its equivalent.

The tuition is $150.00, $50.00 payable on application, the remaining $100.00 to be paid at the time of registration. Arrangements have been made for the participants to be housed in the Nurses Home and to have their board at the University Hospitals. The cost for room, board, and laundry will be approximately $75.00 per month per person.

In order to be eligible to take the examination leading to certification by the American Orthoptic Council, a candidate must have, in addition to the basic course sponsored by the council, or an equivalent course, a minimum of seven months of practical training under a certified orthoptic technician in an office or institution approved by the council. The council makes every effort to find suitable positions for training for all participants in the course who desire one.

For further information about the course or to apply for admission write to:

Hermann M. Burian, M.D.
Chairman, Committee on Instruction
University Hospitals
Iowa City, Iowa

The Purpose of the Guild

- The aim of the Guild of Prescription Opticians of America is to advance the science of ophthalmic optics through the development of a country-wide ethical optical dispensing service that comprehensively meets the needs of the Eye Physicians and their patients; and to educate the public to the fact that the Eye Physician-Guild Optician type of eye service truly renders the most desirable form of eye care.

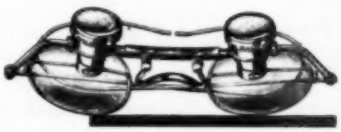
This statement of the Guild's purpose is reprinted from the cover of the REFERENCE LIST OF GUILD OPTICIANS, a geographical index of all our members. If you would like a copy, just drop a penny post card to

The Guild of Prescription Opticians of America, Inc.
110 E. 23rd Street New York (10) N.Y.

E. B. Meyrowitz

SURGICAL INSTRUMENTS CO., INC.,
520 FIFTH AVENUE, NEW YORK 36, N. Y.

LONDON ESTABLISHED 1875 PARIS



ZEISS LOUPE Made by Carl Zeiss, Jena

Gives magnified stereoscopic vision combined with long working distance for detailed observations, examinations and operations.

Lightweight and comfortable, the Gullstrand Magnifying Spectacles provide a magnified image free of distortion and aberration over the entire field of view. With a long working distance and a wide field, the spectacles are extremely well adapted for detailed observation of every description. While working, the wearer can look past the magnifiers to obtain an unobstructed view of his surroundings.

Precision made in the finest Zeiss tradition, the spectacles have full interpupillary adjustment and may be worn in conjunction with spectacle lenses for persons with defective vision.

Supplied in case, without eye wires **$67.50**
As illustrated, but without prescription lenses **72.50**

ISO-SOL®

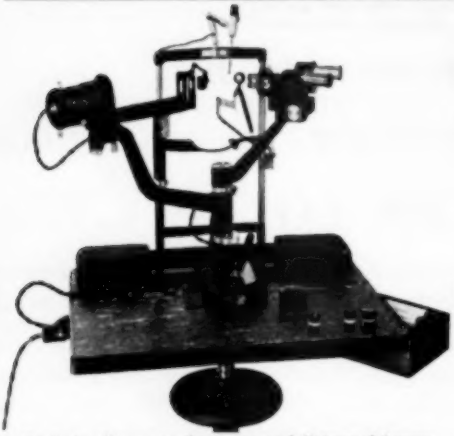
SUPERIOR SOLUTIONS OF MIOTICS
FOR IMPROVED GLAUCOMA THERAPY

1. **Isoosmotic with tears.**
2. **Maximum physiologic activity.**
3. **Notable comfort, minimal irritation.**
4. **Sterile, preserved against contamination.**

Pilocarpine HCl. ½%, 1%, 2%, 4%
Eserine Sal. ⅛%, ¼%, ½%
Pilocarpine HCl. 2% plus Eserine Sal. ¼%*
Pilocarpine HCl. 2% plus Eserine Sal. ½%*

* Supplied in 15 cc sealed sterile dropper bottles.

THE ISO-SOL CO., Inc.
130 Flatbush Ave., Brooklyn 17, N. Y.



Trade in Allowance for Poser and Universal Lamps

THE UNIQUE

GOLDMANN SLIT LAMP

- **One Arm Control**
- **Hruby Lens for Fundus Examination**
- **Many Other Advantages**

U.S. Agents Also for:

Perimeters, Ophthalmometers and Other Ophthalmological Equipment.

ALFRED P. POLL

Dispensing Opticians

40 WEST 55TH STREET, NEW YORK 19, N.Y.

ARTIFICIAL EYES
MADE TO ORDER
AND FITTED EXPERTLY

GREINER & MUELLER

GLASS & PLASTIC

55 E. Washington St. . . . Chicago 2, Ill.
Phone FR 2-4449
Branches at Kansas City, Mo., Detroit, Mich.
Our experts visit Milwaukee, Minneapolis and St. Louis regularly. Serving the Middle West since 1924.

Eye making has been a family tradition with us since 1835

The "Lacrilens"® is a Safe Lens

1. Free From Danger of Injury to The Eyes.
2. Will Not Fall Out of The Eye.
3. Safe For All Sports, Including Swimming.
4. Custom Made to Fit The Eye.

WRITE FOR BROCHURE

Obrig Laboratories Inc.

49 EAST 51st STREET • NEW YORK 22, N.Y.

Mfgs. of all Types of Contact Lenses

BRANCHES IN
PHILADELPHIA . . . MONTREAL . . . LONDON
JOHANNESBURG . . . SHANGHAI

Obrig®

COMPLETE OPHTHALMIC SERVICE FOR THE PROFESSION

Benson OPTICAL COMPANY

Laboratories in Minneapolis and Principal Cities of Upper Midwest

Since 1913

"OPHTHALMIC LITERATURE"

AN ABSTRACT JOURNAL IN ENGLISH OF THE WORLD'S CURRENT LITERATURE ON OPHTHALMIC SUBJECTS

Indispensable to the busy practitioner for quick reference and timely useful information.

"Ophthalmic Literature" and Index (8 numbers) Yearly subscription—Four Guineas ($13.50 U.S.A.). Published by British Medical Association.

U. S. A. Agent

GRUNE & STRATTON, INC.
381 Fourth Ave.
New York 16, New York

the cosmetic
STENOPEIC SPECTACLE

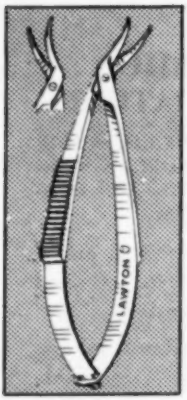
A PRACTICAL AND COSMETIC VISUAL AID WITH MANY USES

- In irregular astigmatism, conical cornea, discrete corneal opacities and incipient cataract the pinholes often permit a clarity not obtainable by spectacle lenses.
- As a temporary expedient the pinholes are valuable for children with high astigmatism (myopic, hyperopic or mixed) until they become sufficiently cooperative for precise objective refraction.
- Following cycloplegia, the pinhole provides protection from glare and compensates for the disturbed visual acuity.
- In pathologic photophobia the pinholes frequently reduce light to a comfortable degree and yet permit effective vision.
- In cases of retinal separation, the motion of the eyeballs can be controlled adequately by blocking all but the central pinholes.
- Used in patients with cataract extraction, before the procurement of their new glasses, the pinhole spectacles add an element of protection besides their other advantages.
- The Cosmetic STENOPEIC Spectacles have many other uses. Write for further information.

• SPECIAL PRICES TO OCULISTS •

SOLD ON A 30-DAY MONEY BACK GUARANTEE IF NOT SATISFIED

SUNSET GLARE GUARD CORP. 1217 WEST GLENOAKS BLVD., GLENDALE 1, CALIFORNIA

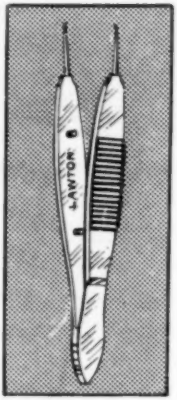


Castroviejo
corneal scissors, for conjuctival dissection especially during cataract extraction. Curved right and left. Blunt points.
Stainless Steel
$25.00 each

Lawton eye instruments
are made exclusively in Germany under conditions most favorable to precision workmanship. Each instrument bearing the Lawton trademark reflects the skill and knowledge of generations of expert instrument makers.

The Lawton Company offers a complete assortment of correct, new patterns, as requested by leading American surgeons. These instruments are sold through authorized surgical supply houses carrying representative stocks, and are thus made available throughout the country for your selection and immediate service.

Visit your local dealer for further information about Lawton instruments. He is well equipped to give you prompt and courteous service.



Castroviejo
suture forceps for corneal scleral and cataract surgery. Regular and delicate model available.
Stainless Steel
$10.00 each

The LAWTON *Company*
425 FOURTH AVENUE, NEW YORK 16, N. Y.

POSTGRADUATE CONFERENCE IN OPHTHALMOLOGY

UNIVERSITY OF MICHIGAN MEDICAL SCHOOL

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the annual conference in Ophthalmology for qualified physicians, April 20, 21, and 22, 1953, to be given at the Horace H. Rackham Graduate School Building, Ann Arbor, Michigan, under the direction of the Department of Ophthalmology at the University of Michigan Medical School.

GUEST LECTURERS

Dr. F. Phinizy Calhoun, Jr., Atlanta, Georgia; Dr. Isadore Givner, New York, New York; Dr. David O. Harrington, San Francisco, California; Dr. M. Noel Stow, Washington, D.C.; Dr. Walter Z. Rundles, Jr., Flint, Michigan.

RESIDENT LECTURERS

Dr. Harold F. Falls, Dr. William H. Havener, Dr. John Woodworth Henderson, Dr. Harry B. McGee, M.D., Dr. Stanley P. Oleksy, Dr. James R. Quinn.

Complete program and details will be mailed upon request addressed to:

HOWARD H. CUMMINGS, M.D., *Chairman*
Department of Postgraduate Medicine
University Hospital Ann Arbor, Michigan

THE WASHINGTON UNIVERSITY SCHOOL OF MEDICINE

DEPARTMENT OF OPHTHALMOLOGY

announces its

THIRTY-THIRD ANNUAL

EIGHT-MONTH BASIC COURSE IN OPHTHALMOLOGY

Didactic, Laboratory, Clinical

September 14, 1953 through May 14, 1954

Class limited to 12 students
Tuition: $800.00

For more detailed information, write to

Lawrence T. Post, M.D.
640 S. Kingshighway Boulevard
Saint Louis 10, Missouri

THE OPHTHALMOLOGICAL STUDY COUNCIL
LANCASTER COURSES IN OPHTHALMOLOGY

Colby College, Waterville, Maine—June 22 to Sept. 5, 1953

SUBJECTS INCLUDED

Anatomy	Bacteriology	Pharmacology	Perimetry
Histology	Optics	Neuro-Ophthalmology	Surgical Principles
Embryology	Physiological Optics	Motor and Sensory	Glaucoma
Heredity	Visual Physiology	Refraction	General Diseases and
Pathology	Bio-Chemistry	Slit Lamp	Ophthalmoscopy

Fee: $325.00. Veterans' Tuition Paid by Veterans Administration
Adequate living quarters on the college campus
For further information write Ophthalmological Study Council
243 Charles Street, Boston 14, Massachusetts

THE TRANSACTIONS
Of the IV Pan-American Congress of Ophthalmology
Mexico 1952

Three volumes comprising a total of 2187 pages in Spanish, Portuguese, and English, with many illustrations, some in color, are now available for distribution.

The complete reports and scientific papers of this noteworthy event.

Apply to: Dr. Thomas D. Allen, 122 So. Michigan Ave., Chicago, Illinois.

Price: three volumes $25.00.

BASIC COURSE IN ORTHOPTICS FOR TECHNICIANS

Sponsored by the
American Orthoptic Council

June 15 to August 15, 1953

At the Department of Ophthalmology
College of Medicine
State University of Iowa

Didactic Lectures and Practical
Demonstrations by
Outstanding Faculty

For Applications and further information write to

HERMANN M. BURIAN, M.D.
Department of Ophthalmology
University Hospitals
Iowa City, Iowa

Opening for young Ophthalmologist in thriving community in Southern New Hampshire, near Boston, Mass. Location on Main Street has been an established practise for fifty years. Over nine thousand patient records. If interested, please write

Mrs. Charles H. Babbitt
277 Main St.
Nashua, New Hampshire

THE "AMIC" MACULA DEFICIENCY TESTER

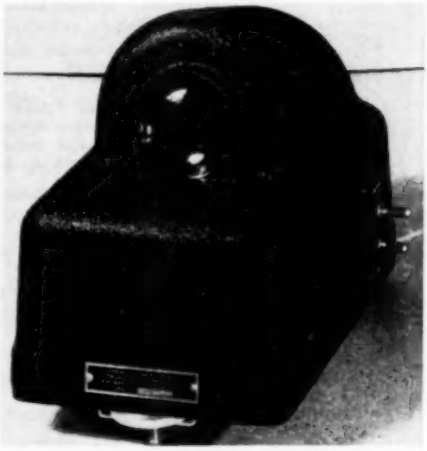
developed by Dr. M. Goldschmidt,
formerly Professor of Ophthalmology, Leipzig University

An instrument to detect early abnormalities of the macula lutea—in a short routine check-up.

As a new instrument of diagnosis, it should belong to the standard equipment of every ophthalmologist.

Write for further information

Recently made available for immediate delivery



AMERICAN MEASURING INSTRUMENTS CORPORATION
21-25 44th Ave., Long Island City 1, N.Y.

•

It Will Pay You

to look through the advertising pages of this Journal.

We discriminate as to the quality and reliability of the advertising accepted for the

AMERICAN JOURNAL *of* OPHTHALMOLOGY

•

THE BRITISH JOURNAL OF OPHTHALMOLOGY

Published monthly by

The British Medical Association

Annual Subscription $13.50

—— • ——

OPHTHALMIC LITERATURE

A comprehensive quarterly abstract of ophthalmology and cognate literature.

Annual Subscription $13.50

—— • ——

Subscriptions to:

GRUNE AND STRATTON, INC.

381 Fourth Avenue

New York 16

New York, U.S.A.

ANNALES D'OCULISTIQUE

One of the oldest ophthalmological reviews. Founded in 1838 by Florent Cunier, continued by Warlomont (1853), Valude and Sulzer (1891), V. Morax (1898).

The Board of Editors is as follows:

REDSLOB	ROCHON-DUVIGNEAUD	MAGITOT
BAILLIART	JEANDELIZE	HARTMANN
HAMBRESIN	LEPLAT	FRANCESCHETTI
AMSLER	P.-V. MORAX	PAUFIQUE
DIAZ-CANEJA	FRANÇOIS	MAGGIORE

Editor-in-Chief: Dr. A. MAGITOT 9 R. de MARIGNAN (Paris)

The publication includes original articles, notes on practical ophthalmology, descriptions of clinical cases and reports of the proceedings of European ophthalmological Societies.

This review is published by Messrs. Doin & Co.
8 Place de l'Odeon Paris 6e

Subscription rates: France and Colonies—4000 Frc.
Abroad —4200 Frc.

Ophthalmologica

International Journal of Ophthalmology
Journal international d'Ophtalmologie
Intern. Zeitschrift für Augenheilkunde

Organ der Schweizerischen Ophthalmologischen Gesellschaft - Organe de la Société Suisse d'Ophtalmologie. Organ van het Nederl. Oogheelkundig Gezelschap - Organ of the Netherl. Ophthalm. Society

Editores:

Aegyptus:
M. Sobby Pasha
R. P. Wilson*

Africa Meridionalis:
A. Jokl
R. C. J. Meyer*
J. S. du Toit

America:
F. H. Adler
H. Barkan
E. V. L. Brown*
J. S. Friedenwald
P. Heath
Bertha Klien
A. C. Krause
B. Samuels
Ph. Thygeson
F. H. Verhoeff
A. C. Woods
A. M. Yudkin

Argentinia:
F. Belgeri*
B. Courtis
M. Dusseldorp
R. Gil
G. v. Grolman

Australia:
J. R. Anderson*
J. A. Flynn

J. B. Hamilton
M. Schneider

Belgica:
M. Appelmans*
L. Coppez
L. Hambresin
A. van Lint
A. van der Straeten
L. Weekers

Brasilia:
M. Alvaro*
C. de Andrade
I. Correa-Meyer
P. Pimentel
L. Silva

Britannia:
A. J. Ballantyne*
St. Duke-Elder
C. B. Goulden
Ida Mann
W. C. Souter

Chile:
C. E. Luca*

Dania:
H. Ehlers
H. Rønne*

Finlandia:
Hilja Teräskeli
M. Vannas*

Gallia:
E. Aubaret
P. Bailliart*
R. Bidault
J. Bollack
P. Bonnet
C. Bourdier
Jean-Gallois
G. Jayle
P. Jeandelize
H. Lagrange
P. Lemoine
J. Mawas
J. Nordmann
R. Onfray
L. Paufique
E. Redslob
Jean-Sédan*
G. P. Sourdille
R. de Saint Martin
Mme S. Schiff
Ch. Thomas
G. Valois
E. Velter
G. Weill

Graecia:
B. Adamantiades
J. Bistis*
J. Charamis
G. F. Cosmetatos
N. Dascalopoulos
C. A. Gabriéldès
Th. Tjanidis
A. Trantas
N. G. Trantas

Helvetia:
M. Amsler
A. Franceschetti
H. Goldmann
E. B. Streiff

Hispania:
H. Arruga*

Hollandia:
H. M. Dekking
J. van der Hoeve
A. W. Mulock Houwer
G. F. Rochat

Hungaria:
G. Ditroi
St. v. Gròsz
G. Horay
A. Kettesy
T. Nónay

India Orient. Neerlandica:
J. G. van Manen*

Italia:
B. Alajmo
G. Lo Cascio
Q. di Marzio*

Luxemburgia:
A. Faber

Ch. Israel
A. Ketter
G. Schintgen

Palaestina:
A. Feigenbaum*

Polonia:
W. Kapuscinski

Portugal:
A. L. de Andrade
S. Senna

Romania:
N. Blatt

Tsechoslovacia:
R. Kadlický
J. Kublik

Tunesia:
R. Nataf*

Turcia:
N. I. Gözoü

Uruguay:
V. Barrière*

* Editor principalis

Redactores: A. BRÜCKNER-Basel, H. WEVE-Utrecht

It is the task of "Ophthalmologica" to advance our knowledge in Ophthalmology by stimulating international co-operation. We publish:

1. Papers in English, German and French.
2. Reviews. One or more subjects of Ophthalmology will be reviewed every month, so that the reader will obtain a comprehensive survey of all the publications which have appeared during the past year.
3. Notes on practical questions. These columns are to be devoted to short interesting observations on cases occurring in everyday practice. They will appear in concise form, perhaps accompanied by an illustration.
4. Reports about the activities of ophthalmological societies.
5. Book Reviews. News.

2 volumes of 6 parts each are published yearly. Subscription price U.S. $10 per volume plus $.60 postage

S. KARGER PUBLISHERS, BASEL, SWITZERLAND

For U.S.A.: Albert J. Phiebig
545 Fifth Avenue, New York 17, New York

A subscription to the Journal would be a most welcome gift to a friend.

Fill in the form at the bottom of this page and mail with your check (rates: domestic $12.00; Canadian and foreign $14.00) to Ophthalmic Publishing Company, 664 North Michigan Avenue, Chicago 11, Illinois.

Please send the American Journal of Ophthalmology for one year to the following residents and students

Name .....Name .....

Address .....Address .....

....

Name .....Name .....

Address .....Address .....

....

Sign Gift Card *from* .....

Your Address .....

(For our records)

Stereoscopic Cards

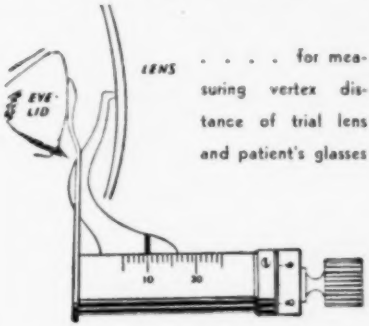
by GUIBOR

Can be used with the Stereocampimeter, Synoptoscope, Rotoscope and similar instruments; also with regular stereoscope. For adults and children.

Set $4.75

For Trial Frame
The Lenscorometer

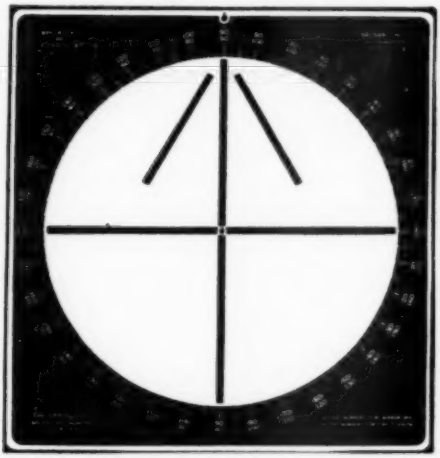
originated by Austin Belgard



. . . . for measuring vertex distance of trial lens and patient's glasses

Use of Lenscorometer in all cases of aphakia, + OR − corrections of four diopters or more—a necessity to insure true translation of prescription.

Each $11.75



(Actual size 13½" x 14")

SIMPLIFIED ASTIGMOMETER

(Lebensohn)

PLASTIC

WASHABLE

"NO COMPUTATION REQUIRED"

Send for Literature

EACH $9.75

OPHTHALMIC OPTICIANS

WHOLESALE & SERVICE

Austin Belgard INC.

109 N. Wabash, at Washington (Formerly Belgard, Inc.) **9th Floor** **STate 2-5362**

AO MONOPLEX makes available...

ALL-PLASTIC EYES

STOCK... alterable at any time for comfortable fit... extremely natural in appearance.

MODIFIED... stock eyes, but with iris colors or veining prepared to suit prescription.

CUSTOM... Completely hand-made to duplicate any eye or combination of eye details.

ORBITAL IMPLANTS

INTEGRATED... plastic bodies with means of muscle attachment. Five different types . . . available in methyl-methacrylate or Kel-F . . . with recess to take peg for Monoplex Eyes

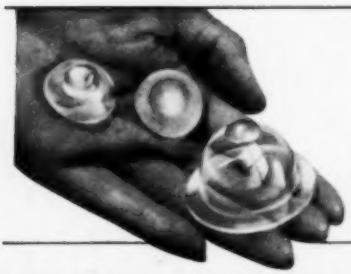
BURIED MESH... Muscles attach to tantalum mesh over round or flattened top... Monoplex Eyes easily fitted. Operative Procedures available.

SPHERES... in clear lucite, stocked in sizes 14 mm., 16 mm., 18 mm. Other sizes available. on special order.

round top

flat top

sphere



CONTACT LENSES

Conventional type lenses . . . made from casting of eye . . . custom-molded to precise specifications with superior optics.

AO Monoplex Laboratory facilities are available for the production of specially designed plastic Implants, Conformers, Lenses, or other plastic devices or components. For details, please call your nearest AO Branch Office.

American Optical